CLINICAL PROFILE OF LEAN BODY WEIGHT TYPE2 DIABETES MELLITUS PATIENTS IN COMPARISON WITH NORMAL WEIGHT AND OBESE TYPE2 DIABETES MELLITUS PATIENTS

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BONAFIDE CERTIFICATE

This is to certify that "CLINICAL PROFILE OF LEAN BODY WEIGHT TYPE 2 DIABETES MELLITUS PATIENTS IN COMPARISON WITH NORMAL WEIGHT AND OBESE TYPE 2 DIABETES MELLITUS PATIENTS" is a bonafide work done by Dr. S.M. SHAVANA**,** post graduate student, Department of General Medicine, **K.A.P. VISWANATHAM** GOVERNMENT MEDICAL COLLEGE, TRICHY-1 under my guidance and supervision in partial fulfillment of regulations of **The Tamilnadu Dr. M.G.R. Medical University for the award of M.D. Degree Branch I, (General Medicine)** during the academic period from May 2008 to April 2011.

Prof. Dr. A. KARTHICKEYAN, M.D.,
DEAN
K.A.P.V. Government Medical College
Trichy -1

Prof. Dr.C.ASOK KUMAR M.D.,
Associate Professor, Unit-II,
Dept of Medicine,
K.A.P.V. Government Medical College
Trichy -1

Prof. Dr. S.PANNEER SELVAM, M.D.,
Professor & Head of the Department,
Dept of Medicine,
K.A.P.V. Government Medical College
Trichy -1

DECLARATION

I Dr. S.M. SHAVANA solemnly declare that the dissertation titled, "CLINICAL PROFILE OF LEAN BODY WEIGHT TYPE 2 DIABETES MELLITUS PATIENTS IN COMPARISON WITH NORMAL WEIGHT AND OBESE TYPE 2 DIABETES MELLITUS PATIENTS" is a bonafide work done by me at Annal Gandhi Memorial hospital affiliated to K.A.P.V. Government Medical College, Trichy-1 under the guidance and supervision of Prof. Dr. S. PANNEER SELVAM, M.D., HOD/Prof of medicine and Unit Chief, Prof. Dr. C. ASOK KUMAR, M.D., The dissertation is submitted to **The Tamilnadu Dr.M.G.R.Medical University,** towards the partial fulfillment of requirement for the award of **M.D degree (Branch-I) in General Medicine.**

Place : Trichirappalli Dr. S.M. SHAVANA

Date :

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CONTENTS

INTRODUCTION

INTRODUCTION

Diabetes Mellitus is a group of metabolic disorders characterized by a deficiency of insulin secretion and / or insulin effect, which causes hyperglycemia, disturbances of carbohydrate, fat and protein metabolism and a constellation of chronic complications. Diabetes is and will remain a threat to global health .World wide diabetes probably affects 150 million people and its prevalence is predicted to double by 2015. The incidence of diabetes is showing an alarming rise in developing countries, particularly in India[3]. 60-80% of the diabetics in developed countries are obese. Whereas in India we find that clinical profile of diabetics is different[1].

Most of the patients attending our diabetic clinic are not obese as defined by existing parameters such as BMI. It is interesting to note that most patients fall in normal weight group and some even lean group. Obesity in type 2 diabetes is less common in Indian population compared to western population[1,4]. So, it is worth studying the clinical profile of lean type 2 diabetes, by comparing with normal and obese population with type 2 diabetes.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Diabetes Mellitus comprises a group of metabolic disorders that share the phenotype of hyperglycemia due to absolute or relative deficiency of insulin. Several distinct types of Diabetes Mellitus exist and are caused by a complex interaction of genetics, environmental factors and life style choices. Lack of insulin affects the metabolism of carbohydrates, protein and fat and causes a significant disturbance of water and electrolyte homeostasis. Though acute metabolic decompensation is fatal, long standing metabolic derangement is frequently associated with permanent and preventable functional and structural changes in the cells of the body, with those of the vascular system being particularly susceptible[5,6]. These changes lead to the development of well defined clinical entities the so called complications of diabetes which characteristically affect the eye, kidney and the nervous system.

Classification[2]

Although all forms of DM are characterised by hyperglycemia the pathogenic mechanisms by which hyperglycemia arises differ widely. Some forms of DM are characterised by an absolute insulindeficiency or

a genetic defect leading to defective insulin secretion, whereas other forms share insulin resistance as their underlying etiology.

ETIOLOGIC CLASSIFICATION OF DIABETES MELLITUS[32]

I. Type 1 diabetes

A. Immune mediated

B. Idiopathic

II. Type 2 diabetes

III. Other specific types

A. Genetic defects of β - cell function

B. Genetic defects in insulin action

C. Diseases of the exocrine pancreas

D. Endocrinopathies

E. Drug - or chemical induced

F. Infections

G. Uncommon forms of immune-mediated diabetes

H. Other genetic syndromes sometimes associated with diabetes

Other types of Diabetes Mellitus[2]

Other etiologies of Diabetes Mellitus include specific genetic defects in insulin secretion or action, metabolic abnormalities that impair insulin secretion, and a host of conditions that impair glucose tolerance. Maturity onset diabetes of the young (MODY)[2] is a subtype of Diabetes Mellitus characterised by autosomal dominant inheritance, early onset of hyperglycemia and impairment in insulin secretion. Mutations in the insulin receptor cause a group of rare disorders characterised by severe insulin resistance. Diabetes Mellitus can result from pancreatic exocrine disease- pancreatitis, fibrocalculous pancreatopathy, haemochromatosis ,etc when the majority of pancreatic islets (>80%) are destroyed.

Endocrinopathies such as Acromegaly and Cushing's disease also, present with Diabetes Mellitus.Rarely viral infections such as rubella, coxsackie and cytomegalo viruses have been implicated in pancreatic islet cell destruction.Drugs and Chemicals such as Glucocorticoids, immunosuppressives, chemotherapeutic agents, B-blockers, thiazides, pentamidine,vacor also play a role in the causation.

Gestational diabetes mellitus

Insulin resistance related to the metabolic changes of late pregnancy increases insulin requirements and may lead to hyperglycemia

or impaired glucose tolerance.These women have a substantial risk of developing Type 2 diabetes in later life.

Lean body type 2 diabetes mellitus patients have severe basal hyperglycemia with low circulatory levels of insulin while C-peptide levels are similar to those of patients with classic type 2 Diabetes mellitus. Studies on hepatic glucokinase levels & hepatic microsomal enzymes (mixed function oxidase & cytochrome P 450) using antipyrine & lidocaine as in vivo probes revealed hyperactivity with increased futile cycles of CHO metabolism in lean body type 2 patients. These hepatic metabolic features are likely to be responsible for excess insulin utilization & extraction during first pass in the liver leading to low peripheral circulating levels. Homocysteine levels are also low suggesting efficient metabolic status[12,13].

Auto immune destruction[11] of beta cells is not the cause of hypoinsulinemia as levels of ICA-512 / IA2 (islet cell antibody)and anti-GAD (anti glutamic acid decarboxylase) antibodies are similar to those in patients with classical type 2 diabetes mellitus & much lower than those in type1 diabetes mellitus. In this way, it differs from the latent auto immune diabetes in aduls (LADA) which present at a later age.

The metabolic profile reveals normal HDL cholesterol levels[9,10] with typeIV hyperlipoproteinemia in glycemic uncontrolled states.

Proteinuria found in uncontrolled metabolic states often reverses suggesting endothelial cell dysfunction.

Lean body type 2 diabetes mellitus is not mere anthropometric variant of classical type 2 diabetes mellitus but constitute an independent variant of type 2 diabetes mellitus[7,8,14] with inherent peculiarities in insulin kinetics in the hepatic bed along with altered profile & behaviour of key enzymes related to CHO metabolism. These peculiarities are reflected in the peripheral circulation as states of hypoinsulinemia, hyperglycemia, dyslipidemia[12,16] without low HDL C, raised TGL & fewer other markers for atherosclerosis which make these diabetics less prone to develop macro vascular disease[14-17], while peripheral neuropathy and consequences of hyperglycemia like infections & proteinuria dominate the clinical picture.

Epidemiology

Diabetes remains a threat to global health. World wide the prevalence of Diabetes is estimated to increase from 4% in 1995 to 5.4% by the year 2025[19]. India has the dubious distinction of having the largest number of diabetics in the world. The prevalence 'of Diabetes in India Study (PODIS) showed that type 2 Diabetes Mellitus was found in 7.06% of the population, which is expected to double by 2015. Diabetes Mellitus

is the leading cause of end stage renal disease, non traumatic lower extremity amputations and adult blindness in U.S. The increasing prevalence of Diabetes Mellitus in developed countries is largely attributed to increasing obesity and reduced activity levels. The prevalence of Type 2 DM and its harbinger, IGT is highest in certain pacific islands, intermediate in countries such as India[1] and United States, relatively less in Russia and China. This variability is likely due to genetic, behavioral and environmental factors. The pattern and profile are very different in India compared to the west[1].

Criteria for the Diagnosis of Diabetes Mellitus[20]

- Symptoms of Diabetes plus random blood glucose concentration > 11.1 mmol/L (200 mg/dl)[(a)] (or)
- Fasting plasma glucose > 7.0 mmol/L (126 mg/dl)[(b)] (or)
- Two hour plasma glucose > 11.1 mmol/L (200 mg/dl) during an oral glucose tolerance test.[(c)]
- HbA1C>/=6.5%[(d)]

a) Random is defined as without regard to time since the last meal.

b) Fasting is defined as no caloric intake for at least 8 hours.

c) The test should be performed using a glucose load containing the equivalent of 75 gm anhydrous – glucose dissolved in water: not recommended for routine clinical use.

d) The test should be performed in a laboratory that is NGSP certified and standardized to the DCCT assay.

Source: Adapted from American Diabetes Association, 2010.

Table 2

Diagnostic Criteria for Pre-Diabetes and Diabetes[20]

Test	IFG	IGT	Diabetes	Gestational Diabetes*
FPG	100-125	Not defined	≥ 126 mg/dl	≥ 95 mg/dl
RPG	Not defined	Not defined	≥ 200 mg/dl	Not defined
75-g OGTT 2-hour plasma glucose	Not defined	140-199 mg/dl	≥ 200 mg/dl	Not defined
100-g OGTT	Not defined	Not defined	Not defined	1-hour: ≥ 180 mg/dl 2-hour: ≥ 155 mg/dl 3-hour: ≥ 140 mg/dl
AIC	Not defined**	Not defined**	≥ 6.5%	Not defined

Adapted from ADA – 2010

PATHOGENESIS[5,6]

Type 1 Diabetes Mellitus

Type 1 Diabetes Mellitus develops as a result of the synergistic effects of genetic, environmental and immunologic factors[5,6] that ultimately destroy the pancreatic beta cells.

1. Genetic Factors

Account for one third of the susceptibility to Type 1 Diabetes, the inheritance of which is polygenic. Over 20 different regions of the human genome show some linkage with type 1 diabetes, but most interest has focused on the human leucocyte antigen (HLA), on the short arm of chromosome 6. The HLA haplotypes DR3 and / or DR4 alleles are associated with increased susceptibility to type 1 diabetes.

2. Environmental factors

Although genetic susceptibility be a prerequisite for the development of type 1 diabetes, the concordance rate between monozygotic twins is less than 40%. Environmental factors have an important role in promoting clinical expression of the disease.

The hygiene hypothesis

Lack of exposure to pathogenic organisms in early childhood limits maturation of the immune system and increases susceptibility to autoimmune disease.

3. Viruses

Several viruses have been implicated, including mumps[5,6], Coxsackie B4, retroviruses, rubella (in utero) Cytomegalovirus and Epstein–Barr virus.

4. Diet

Bovine serum albumin (BSA), a major constituent of cow's milk, has been implicated in triggering type 1 diabetes. It has been shown that children who are given cow's milk early in infancy are more likely to develop type 1diabetes than who are breast fed.

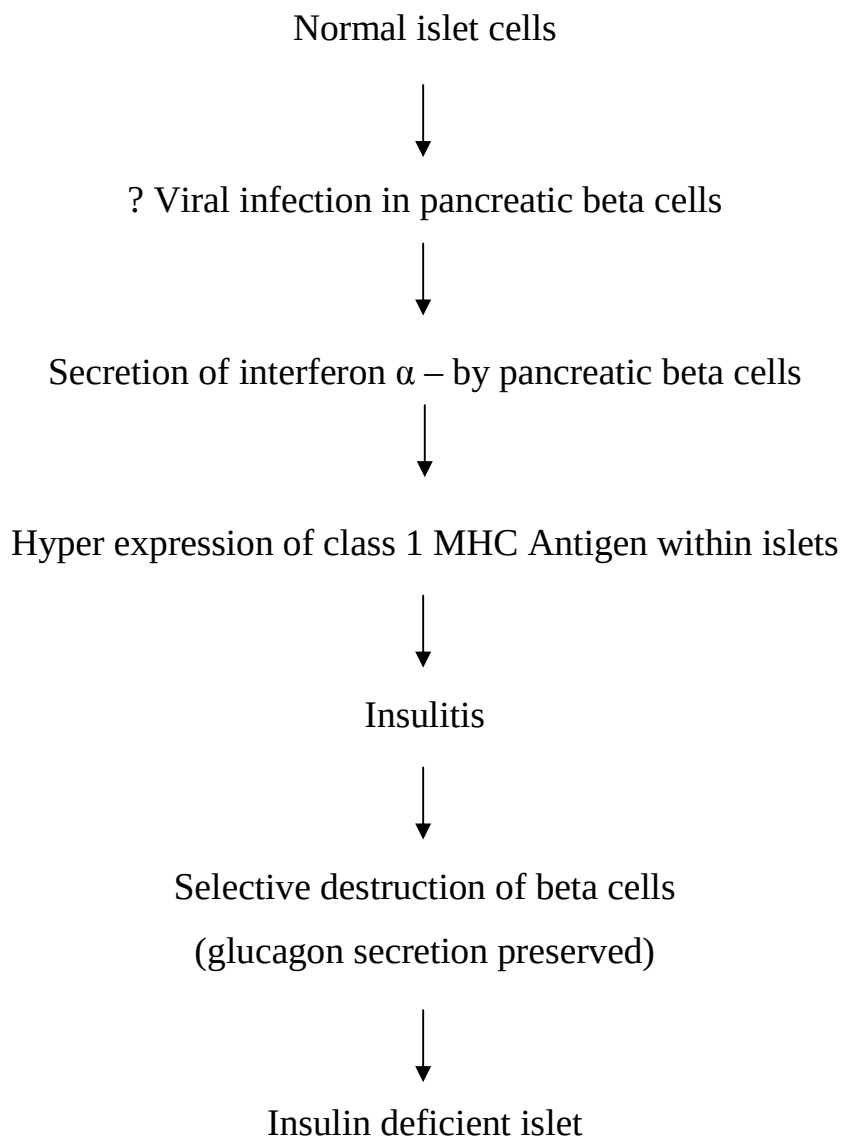
5. Stress

Stress may accelerate the development of type 1diabetes, by increasing counter regulatory hormones and possibly by modulating immune activity.

6. Immunological factors

Type 2 diabetes is a slow T cell mediated autoimmune disease. Many studies have produced evidence that destruction of the insulin secreting cells in the pancreatic islets takes place over many years.

Pathogenesis of Type 1 Diabetes



Type 2 Diabetes Mellitus

Type 2 Diabetes mellitus commonly occurs in subjects who are obese and insulin resistant, but these two factors alone are insufficient to cause diabetes unless accompanied by impaired beta cell function.

1. Genetics

Genetic factors are more important in the etiology of type 2 DM than type 1 diabetes, as shown by studies in monozygotic twins where concordance rates of type 2 diabetes approaches 100%.

2. Environmental Factors

The majority of cases of type 2 diabetes are multifactorial in nature, with interaction of environmental and genetic factors.

a) Life style: Overeating, fastfood eating habits especially when combined with obesity and underactivity.

b) Malnutrition in utero: It is proposed that, (but not yet proven), malnutrition in utero may programme beta cell development and metabolic functions at a critical period, so predisposing to type 2 diabetes later in life.

c) Age: Age is an important risk factor for type 2 diabetes. Type 2 Diabetes is principally a disease of the middle aged and elderly, affecting 10% of the population over the age of 65.

d) Pregnancy: During normal pregnancy, insulin sensitivity isreduced through the action of placental hormones and this affects glucose tolerance.

Pathogenesis of Type 2 Diabetes Mellitus[5,6]

i) Insulin resistance

ii) Pancreatic Beta cell failure

1. Insulin Resistance

Increased hepatic production of glucose and resistance to the action of insulin in muscle are invariable in both obese and non obese patients with type 2 diabetes. Insulin resistance may be due to

a) an abnormal insulin molecule

b) an excessive amount of circulating antagonists or

c) Target tissue defects

The last is the most common cause of insulin resistance in type 2 diabetes.

2. Pancreatic Beta Cell Failure[5,6]

In type 2 DM, there is only moderate reduction in the total mass of pancreatic islet tissue which is consistent with a measurable fall inplasma insulin concentration. Some pathological changes are typical of type 2 diabetes, most conspicuous of which is deposition of amyloid. While beta cell numbers are reduced by 20-30% in type 2 diabetes, alpha cell mass is unchanged and glucagon secretion is increased, which may contribute to the hyperglycemia[5,6].

Some people with type 2 diabetes, most of whom are not overweight, have advanced pancreatic beta cell failure at the time of presentation and require early treatment with insulin.

PATHOGENIC PROCESS OF DIABETES MELLITUS

Type of diabetes	Normal glucose tolerance	Hyperglycemia			
		Pre-diabetes	Diabetes mellitus		
		Impaired fasting glucose or Impaired glucose tolerance	Not insulin requiring	Insulin required for control	Insulin required for survival
Type 1	←	—	—	—	→
Type 2	←	—	—	→	
Others	←	—	—	→	
GDM	←	—	—	→	
Time (years)	—	—	—	—	→
FPG	100mg/dl	100-125%	126 mg/dl		
2-h PPG	140 mg/dl	140-199mg%	200 mg%		

SPECTRUM OF GLUCOSE HOMEOSTASIS AND DIABETES MELLITUS

In most types of DM, the individual traverses from normal glucose tolerance to impaired glucose tolerance to overt Diabetes. In some types, the changes in glucose tolerance may be bidirectional.

COMPLICATIONS OF DM

Acute Complications

Diabetic ketoacidosis (DKA) and Hyperglycemic hyperosmolar state (HHS) are acute complications[2] of diabetes. DKA is seen primarily in individuals with type 1 Diabetes Mellitus, and HHS is seen in individuals with type 2 Diabetes Mellitus. Both disorders are associated with absolute or relative insulin deficiency, volume depletion and altered mental status. Both are potentially serious if not promptly diagnosed and treated. Side effects of intensive treatment include severe hypoglycemia and Lactic acidosis[21].

Chronic complications

Chronic complications[2] of DM affect many organ systems and are responsible for majority of morbidity and mortality.

Chronic complications of Diabetes Mellitus[2]

Microvascular	Macrovascular	Others
Eye disease: Retinopathy	Coronary artery disease	Gastrointestinal
Macular oedema	Peripheral vascular disease	Genito urinary
	Cerebrovascular disease	Dermatological
Neuropathy		Cataract
Sensory and Motor		Glaucoma
Autonomic		Peiodontal disease
Nephropathy		

The risk of complications of both type 1 and type 2 increases as a function of the duration of hyperglycemia. They usually become apparent in the second decade of hyperglycemia.

Mechanism of complications[5,6]

Three major theories have been proposed to explain the emergence of complications.

1. Increased intracellular glucose leads to the formation of advanced glycosylation end products (AGEs) via non enzymatic glycosylation of cellular proteins. AGEs have been shown to cross link proteins, accelerate atherosclerosis, promote glomerular dysfunction, reduce nitric oxide synthesis, induce endothelial dysfunction and alter the extracellular matrix composition and structure.
2. Hyperglycemia increases glucose metabolism via the sorbitol pathway. Increased intracellular glucose is converted to sorbitol by the enzyme aldose reductase. Increased sorbitol concentrations affect several aspects of cellular physiology and may lead to cellular dysfunction.
3. Hyperglycemia increases the formation of diacylglycerol leading to activation of certain isoforms of protein kinase C, which in turn, affect a variety of cellular events that lead to Diabetes Mellitus

related complications. Finally oxidative stress and free radical generation may also promote the development of complications.

Diabetic Retinopathy

Diabetic retinopathy is the most common cause of blindness in adults. Hyperglycemia increases retinal blood flow and metabolism and has direct effects on retinal endothelial cells and pericytes, loss of which impairs vascular auto regulation. The resulting uncontrolled blood flow increases production of vasoactive substances and endothelial cell proliferation resulting in capillary closure. This causes chronic retinal hypoxia and stimulates production of growth factors, including vascular endothelial growth factor (VEGF) to stimulate endothelial cell growth (causing new vessel formation) and increased vascular permeability (causing exudative damage).

Diabetic Nephropathy

Diabetic Nephropathy is the leading cause of end stage renal disease (ESRD) in many countries.

Mechanism of chronic hyperglycemia to ESRD involves

1. interaction of soluble factors (AT II, AGEs, Endothelin)
2. hemodynamic alterations in renal microcirculation.
3. structural changes in the glomerulus.

Diabetic neuropathy

"A descriptive term meaning a demonstrable disorder, either clinically evident or subclinical that occurs in the setting of diabetes mellitus without other causes for peripheral neuropathy. The neuropathic disorder includes manifestations in the somatic and/or autonomic parts of the peripheral nervous system.

Aetiopathogenesis of Diabetic Neuropathy

Hypotheses concerning the multiple etiologies of diabetic neuropathy include a metabolic insult to nerve fibers[22], neurovascular insufficiency, autoimmune damage, and neurohormonal growth factor deficiency. Several different factors have been implicated in this pathogenic process. Hyperglycemic activation of the polyol pathway leading to accumulation of sorbitol and potential changes in the NAD: NADH ratio may cause direct neuronal damage and/ordecreased nerve blood flow (Greene et al, 1983). Activation of protein kinase C induces vasoconstriction and reduces neuronal blood flow (Veves et al, 2001). Increased oxidative stress, with increased free radical production causes vascular endothelial damage and reduces nitric oxide bioavailability (Cameron et al, 1997). Alternatively, excess nitric oxide production may result in formation of peroxynitrite and damage the endothelium and

neurons. In a subpopulation of individuals with neuropathy, immune mechanisms may also be involved. Reduction in neurotrophic growth factors, deficiency of essential fatty acids, and formation of advanced glycosylation end products (localized in endoneurial blood vessels (Brownlee, 1992) also result in reduced endoneurial blood flow and nerve hypoxia with altered nerve function. The result of this multifactorial process may be activation of polyADP ribosylation and depletion of ATP, resulting in cell necrosis and activation of genes involved in neuronal damage.

Diabetic autonomic neuropathy

A subtype of the peripheral polyneuropathies that accompany diabetes, Diabetic autonomic neuropathy (DAN) can involve the entire autonomic nervous system (ANS) the vasomotor, visceromotor, and ensory fibers of which innervate every organ. Diabetic autonomic neuropathy may be either clinically evident or subclinical. It is manifested by dysfunction of one or more organ systems (e.g.,cardiovascular, gastrointestinal, genitourinary, or ocular). Indeed,because the vagus nerve (the longest of the ANS nerves)accounts for roughly 75% of all parasympathetic activity and, Diabetic autonomic neuropathy manifests first in longer nerves, symptoms suggestive of

autonomic dysfunction may be common. They may frequently be due to other causes rather than to true autonomic neuropathy. Subclinical autonomic dysfunction can however, occur within a year of diagnosis in type 2 diabetes patients (Pfeifer et al, 1984). Cardiovascular autonomic neuropathy (CAN) is the most clinically important and well-studied form of Diabetic autonomic neuropathy as it is associated with various adverse outcomes.

Macrovascular Complications[2]

1. Cardiovascular Morbidity and Mortality

Framingham Heart study revealed a marked increase in congestive heart failure, coronary artery disease, myocardial infarction (MI), Peripheral arterial disease and sudden death (risk increases from one to five fold) in DM. American Heart Association recently designated Diabetes mellitus as a major risk factor for cardiovascular disease (same category as smoking, hypertension and hyperlipedemia).

The absence of chest pain (silent myocardial ischemia) is common in individuals with diabetes and a thorough cardiac evaluation is indicated. Coronary artery disease is more likely to involve multiple vessels in individuals with diabetes mellitus[23].

2. Hypertension

Hypertension in diabetes mellitus can accelerate other complications of DM, particularly cardiovascular disease[23], and nephropathy. Blood pressure goal in individual with diabetes is < 130 /80 mm Hg. It is often difficult to control hypertension with a single agent especially in type 2 DM.

3. Dyslipidemia

Individuals with diabetes may have severe forms of dyslipidemia. Because of additive cardiovascular risk of hyperglycemia and hyperlipidemia, lipid abnormalities should be aggressively detected and treated[23,24]. Most common pattern of dyslipidemia is hypertriglyceridemia and reduced HDL cholesterol levels.

Target lipid values[10,20] in diabetic individual without cardiovascular disease should be,

- LDL < 100 mg/dl
- HDL (>40 mg/dl) in men
- HDL (>50 mg/dl) in women
- Triglycerides < 150 ml/dl
- ADA recommends an LDL level of <70 mg/dl in those with cardiovascular disease.

4) Lower extremity complications[2]

Diabetes is the leading cause of non traumatic lower extremity amputations. Foot ulcers and infections are also a major source of morbidity in individuals with DM.

5) Infections[2]

Individuals with DM have a greater frequency and severity of infection. The reasons for this include incompletely defined abnormalities in cell mediated immunity and phagocytic function associated with hyperglycemia, as well as diminished vascularisation. Cardiac and other fungal infections, emphysematous infections of the gall bladder and urinary tract, pneumonia and skin and soft tissue infections are all more common in diabetic population. However gram negative organisms, M.tuberculosis and S. Aureus are also more frequent pathogens.

Diabetic skin complications[2]

1. Diabetic dermopathy – begins as an erythematous area and evolves into an area of circular hyperpigmentation.

2. Necrobiosis Lipoidica – Diabeticorum – usually begins in the pretibial region as an erythematous plaque or papules

that gradually enlarge, darken and develop irregular margins with atrophic centers and central ulceration.

3. Acanthosis nigricans – Hyperpigmented velvety plaques seen on the neck, axilla, or extensor surfaces, is sometimes a feature of severe insulin resistance.

4. Granuloma Annulare – erythematous plaques on the extremities or trunk.

5. Scleredema – Area of skin thickening on the back or neck at the site of previous superficial infections.

6. Lipoatrophy and Lipohypertrophy

7. Xerosis and **pruritus** are common.

CLINICAL PROFILE OF LEAN TYPE 2 DIABETES

Articles review

1. Clinical profile of lean type 2 diabetes – study conducted at Madras Diabetes Research Foundation[14], India with 347 lean, 6274 normal and 3252 obese type 2 diabetes patients in 2002 observed.
    a. 60% are non obese and lean type 2 DM constituted 3.5%.
    b. Increased prevalence of retinopathy, nephropathy and neuropathy in lean type 2 DM patients.
2. Clinical profile of type 2 diabetes mellitus and body mass index[25] – is there any correlation?. Study conducted with 500 patients at Manipal, Kasthurba Medical College by Prabhu Mukhyaprana in 2004 observed,
    a. Majority (65%) belonged to normal weight diabetes group, and 7.1% were lean diabetics.
    b. Most of the lean diabetics were males (65%) with less positive family history.
    c. There was linear increase in number of patients having abnormal WHR with increase in BMI.
    d. Microvascular complications were found in similar proportion in all groups.

e. Lean diabetics are less prone to develop macro vascular complications like HT and IHD.

f. Lean diabetics have more severe hyperglycemia and poor metabolic control.

g. Analysis of lipid profile showed, all the parameters were lower in lean diabetics compared to other groups i.e. normal and obese patients.

3. Clinical profile of lean body weight type 2 DM patients in comparison with obese and non obese type 2 diabetes patients: Study conducted at Jamnagar, M.P. Shah Medical College by Gohel DR, Deszi VK[26], in 2002-2003 observed very similar results as previous studies. In addition,

   i) Increased incidence of higher fasting plasma glucose (239+42.5) in lean diabetics.

   ii) Peripheral neuropathy (52%) and infections (42%) were the commonest presenting clinical features in lean patients[26].

4. Increased prevalence of Retinopathy, nephropathy and neuropathy in lean diabetics; Mohan et al.

5. Studies by Banerji et al and Dass et al had showed slight increase in Triglycerides (TGL) and HDL in lean diabetes.

6. Japanese study by Ikeda et al showed no major differences in lipid profile in lean diabetics irrespective of glycemic status.

One observation from the National Institute of Diabetes and Digestive kidney diseases, Phoenix, that “NIDDM in the presence of low BMI is more strongly familial than that at a higher BMI,” warrants further study into the possible genetic mechanisms that modulate the above factors in Lean Type 2 DM[31].

AIMS AND OBJECTIVES

AIM OF THE STUDY

1. To Study and compare the clinical profile of Lean Body weight Type 2 Diabetes Mellitus patients with obese and Normal weight Type 2 DM patients, by age, sex, family history and Anthropometry.
2. To compare the "presenting complications" of the lean with normal weight / obese type 2 Diabetes patients.
3. To compare the Biochemical profile of the lean type 2 Diabetes with that of normal and obese type 2 DM.

MATERIALS AND METHODS

MATERIALS AND METHODS

Type of the study : Cross sectional

Period of study : January 2010 – September 2010

Place of study : Annal Gandhi Memorial Government Hospital, Trichy.

No.of Patients : 100

Materials : Type 2 Diabetes Mellitus patients

The hundred patients were divided into three groups based on BMI.

Body Mass Index (BMI)

Group A: BMI < 18.5 Kg/m2 (Lean Body Weight Type 2DM)

Group B: BMI, between 18.5 and 24.9 Kg/m2 (Normal Weight Type 2 DM).

Group C: BMI > 30Kg/m2 (Obese Type 2 DM)

A careful detailed history were taken from each person, i.e. Age of onset, duration, any positive family history, dietary pattern, presenting complaints – at the time of diagnosis etc. Detailed examination was done for all the hundred patients to find out various complications, if any. Biochemically, Blood glucose (Both fasting and post prandial), Blood urea, Serum Creatinine, Lipid profile were analysed in all the three groups.

Selection of cases

Cases included in the study were selected as per the records available with them. Duration of disease, Body Mass Index, Waist Hip Ratio, current Blood Glucose, Urea, Serum Creatinine and Lipid profile were taken into consideration.

Exclusion Criteria for cases

1. Presence of history of pulmonary tuberculosis.
2. Presence of other chronic illnesses that could affect body weight like chronic liver disease and chronic kidney disease.
3. Type 2 Diabetes patients with Age of onset less than 30 years.
4. History wise, particularly in lean patients those who were normal or obese at the time of presentation, but lost body weight significantly after the detection of type2 Diabetes mellitus.
5. Patients with history of Cancer /HIV.
6. Over weight patients with BMI between 25-30.

Selection of Controls

Control cases were normal weight as well as obese patients with Type 2 Diabetes Mellitus.

Methods

1. Height (in meter), Weight (in kg) measured in all patients.

BMI (Body Mass Index) calculated based on the formula,

BMI (kg/m2) = Weight (kg)/Height (in m2) (Quetelet)

2. Waist hip Ratio (W/H Ratio)

- 'Waist Circumference' measured at midpoint between the costal margin and anterior superioriliac spine. Hip measurement taken as maximum diameter at the greater trochanter.
- Waist / Hip Ratio (WHR) was calculated in each case.
- Waist Hip ratio was considered abnormal if > 0.9 for males and > 0.8 for females.

Patients were clinically screened for microvascular and macrovascular complications.

- Patients were considered as hypertensives if blood pressure was > 130/80 mm Hg.
- Patients were considered as having ischemic heart disease based on ischemic changes in the ECG or by demonstrating regional wall motion abnormalities in the echocardiogram for selected patients.
- Ophthalmoscopy was done to diagnose diabetic retinopathy. Neuropathy was diagnosed, based on subjective symptoms or

objective evidence in the form of loss of ankle jerk or glove and stocking type of anaesthesia.

- Nephropathy was diagnosed based on blood urea and serum creatinine values and Ultrasound abdomen and urine microalbumin in selected patients.
- Fasting, postprandial glucose, fasting lipid profile and other relevant investigations were done in each case.

Definitions and Cut Off values for the study[28]

1. Body Mass Index (BMI)

18.5-24.9 (kg/m2) – taken as normal value

<18.5 (kg/m2) – lean body weight

>30 (kg/m2) – obese body weight

2. Waist Hip Ratio

WHR - >0.85– was taken as abnormal value in females.

>0.9 in males as abnormal value.

3. Fasting 'Hyperglycemia'

Fasting Hyperglycemia means if Blood glucose value >126 mg% In the fasting state.

4. Post prandial Hyperglycemia

Postprandial blood sugar measured at 2 hours after the meals. Post prandial hyperglycemia means if value > 200 mg%

5. Lipid Profile

Lipid profile taken after 8 hours overnight fasting.

Range of Normal Values

- Free cholesterol < 200 mg/dl
- LDL < 100 mg/dl
- HDL (>40 mg/dl) in men
- HDL (>50 mg/dl) in women
- Triglycerides < 150 ml/dl
- ADA recommends an LDL level of <70 mg/dl in those with cardiovascular disease.

Others

Blood is drawn from each patient under recommended ideal conditions to determine the fasting and postprandial Blood sugar, urea, serum creatinine and Lipid profile.

Ethical Committee Approval

The present study was approved by the ethical committee.

Statistical Analysis

Statistical Analysis of data was done by using the software – Statistical Packages for Social Sciences (SPSS version 13.0) developed by LEAD TOOLS CORPORATION.

RESULTS AND OBSERVATIONS

RESULTS AND OBSERVATIONS

TABLE - 1

CHARACTERISTICS OF THE STUDY POPULATION

S. No	CHARACTERISTICS	RANGE	MEAN	S.D.
1.	AGE	35-65	56.1	8.877
2.	BMI	16-35	2.13	0.0719
3.	WAIST HIPRATIO			
	MALES	0.78-1.0		
	FEMALES	0.78-1.0		
	TOTAL		1.64	0.04842
4.	FASTING BLOOD SUGAR	90-360	1.93	0.06397
5.	POSTPRANDIAL BLOOD SUGAR	190-500	2.39	0.06948
6.	TOTAL CHOLESTEROL	150-290	1.49	0.05024
7.	TGL	40-350	1.48	0.05021
8.	HDL	25-90	1.43	0.04976
9.	LDL	60-210	1.76	0.04292
10.	VLDL	10-140	32.46	2.095
11.	BP: SYSTOLIC	100-170	127.18	2.078
	DIASTOLIC	60-110	82.68	1.262

TABLE – 2

COMPLICATIONS

S. No.	CHARACTERISTICS	NO	%
1	SEX MALES	48	48
	FEMALES	52	52
2.	FAMILY HISTORY YES	31	31
	NO	69	69
3.	COMPLICATIONS		
a.	CARDIAC YES	36	36
	NO	64	64
b.	RENAL YES	25	25
	NO	75	75
c.	NEURO YES	20	20
	NO	80	80
d.	RETINO YES	17	17
	NO	83	83
e.	INFECTIONS YES	17	17
	NO	83	83
f.	HYPERTENSION YES	43	43
	NO	57	57
g.	BMI LEAN	19	19
	NORMAL	49	49
	OBESE	32	32

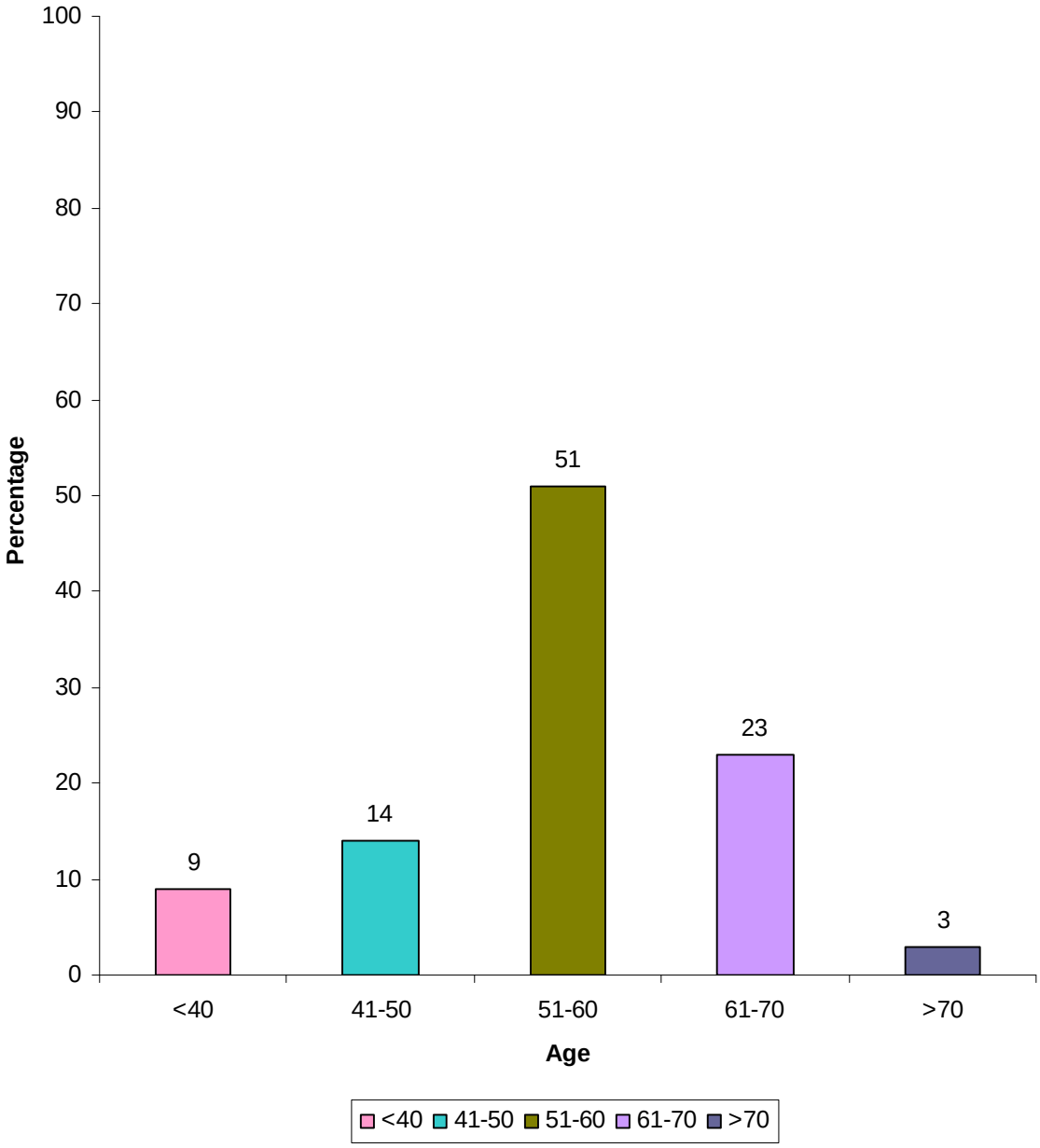
TABLE – 3

AGE AND BMI

AGE GROUP	LEAN		NORMAL		OBESE	
	No.	**%**	**No**	**%**	**No**	**%**
< 40 Yrs	-	-	4	44.4	5	55.6
41-50Yrs	2	14.3	8	57.1	4	28.6
51-60Yrs	12	24.5	24	48.9	13	26.5
> 60Yrs	5	17.8	13	46.4	10	35.7
TOTAL	19		49		32	

There is no statistically significant relationship between age and BMI.

AGE & BMI

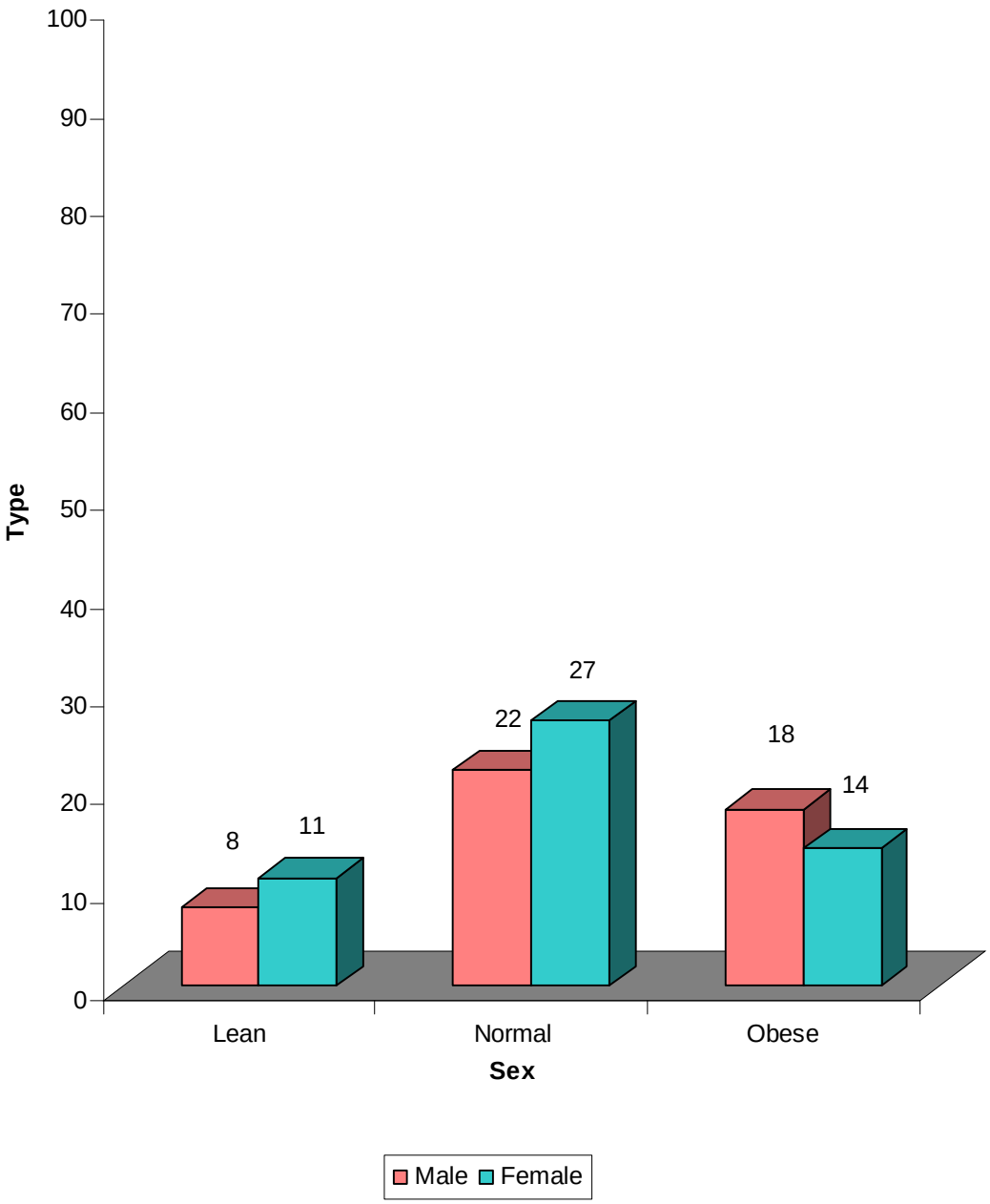

100
90
80
70
60
50
40
30
20
10
0
Percentage
9
14
51
23
3
<40
41-50
51-60
61-70
>70
Age
<40
41-50
51-60
61-70
>70

SEX AND BMI

SEX	TYPE					
	LEAN		NORMAL		OBSES	
	NO	%	NO	%	NO	%
MALE	8	16.6	22	45.8	18	37.5
FEMALE	11	21.2	27	51.9	14	26.9
TOTAL	19		49		32	

There is statistically significant relationship between sex and BMI.

SEX AND BMI


100
90
80
70
60
50
40
30
20
10
0
Type
8
11
22
27
18
14
Lean
Normal
Obese
Sex
Male
Female

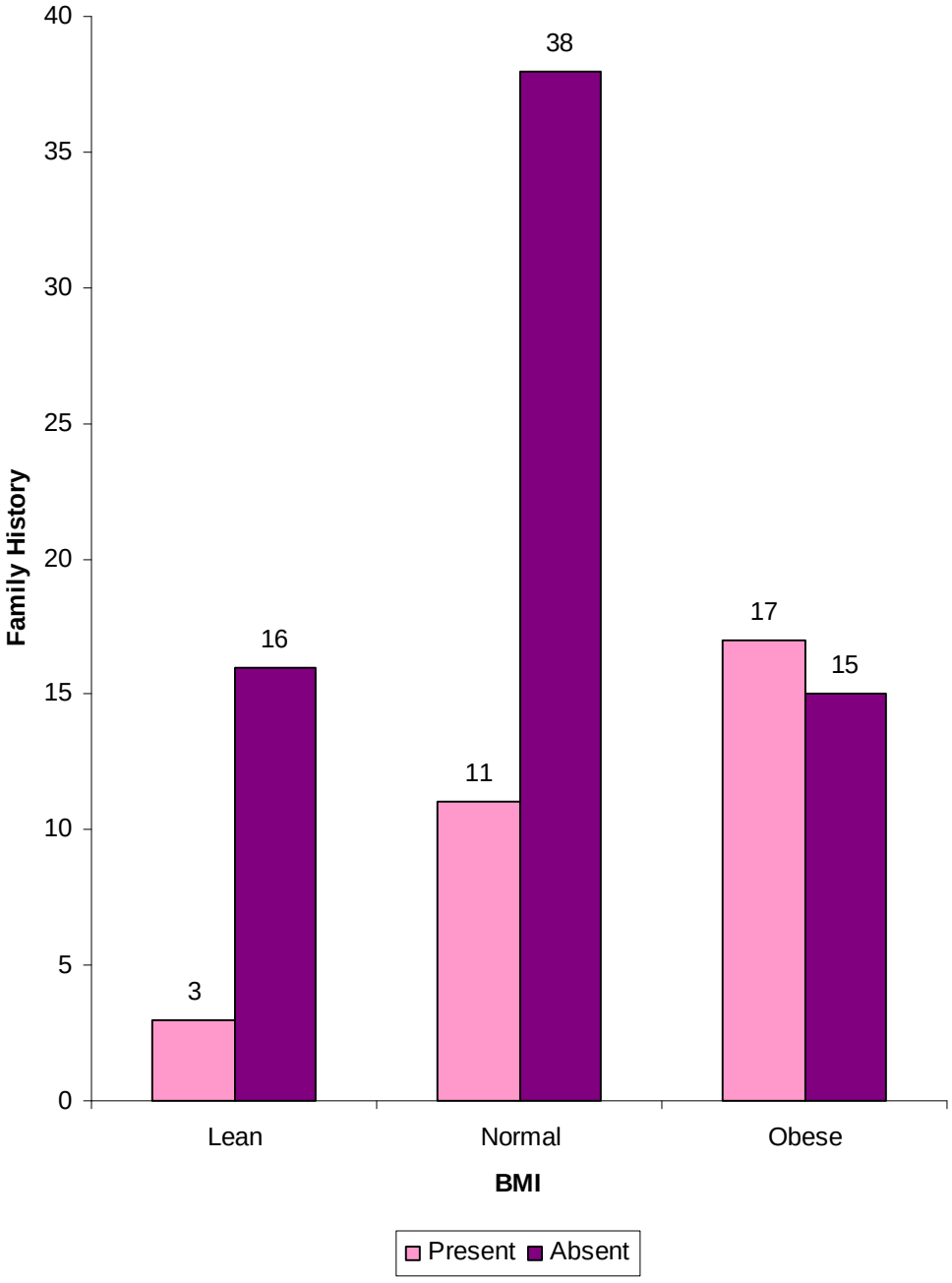
FAMILY HISTORY AND BMI

FAMILY HISTORY	LEAN		NORMAL		OBSE	
	NO	%	NO	%	NO	%
YES	3	9.6	11	35.4	17	54.8
NO	16	23.1	38	55.1	15	21.7
TOTAL	19		49		32	

Statistically significant at 0.01 level.

% of lean cases is low in persons with family history.

FAMILY HISTORY AND BMI



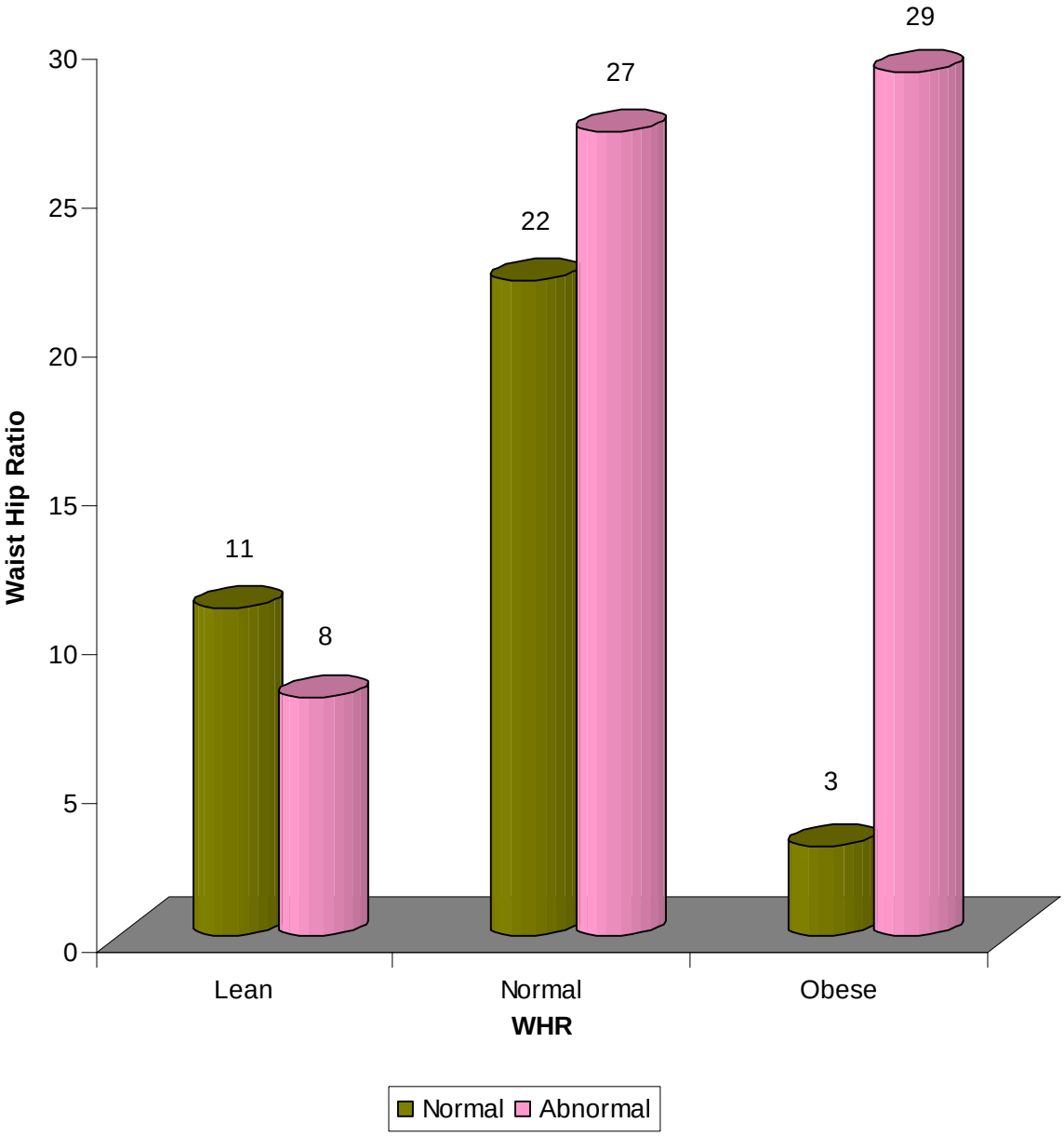
WAIST HIP RATIO AND BMI

WHR	LEAN		NORMAL		OBESE	
	NO	%	NO	%	NO	%
NORMAL	11	30.5	22	61.1	3	8.3
ABNORMAL	8	12.5	27	42.2	29	45.3
TOTAL	19		49		32	

There is statistically significant relationship between WHR and BMI.

'p' value 0.01.

WAIST HIP RATIO AND BMI



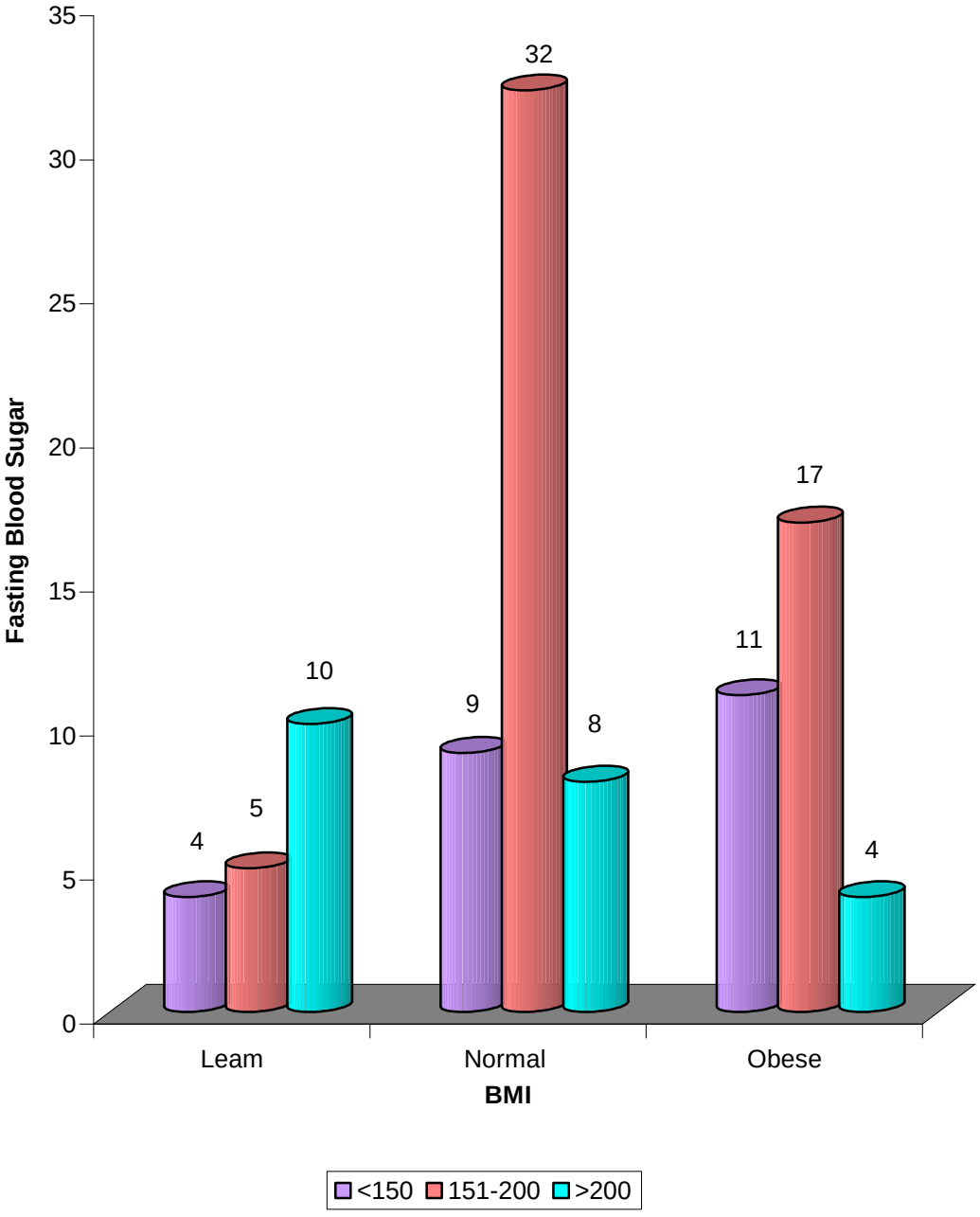
FASTING BLOOD SUGAR AND BMI

FASTING BLOOD SUGAR	LEAN		NORMAL		OBESE	
	NO	%	NO	%	NO	%
NORMAL	4	16.7	9	37.5	11	45.8
ABNORMAL	15	19.7	40	52.6	21	27.6
TOTAL	19		49		32	

There is statistically significant relationship between Fasting blood sugar and BMI.

'P': -0.112 Significant at 0.05 level

FASTING BLOOD SUGAR AND BMI

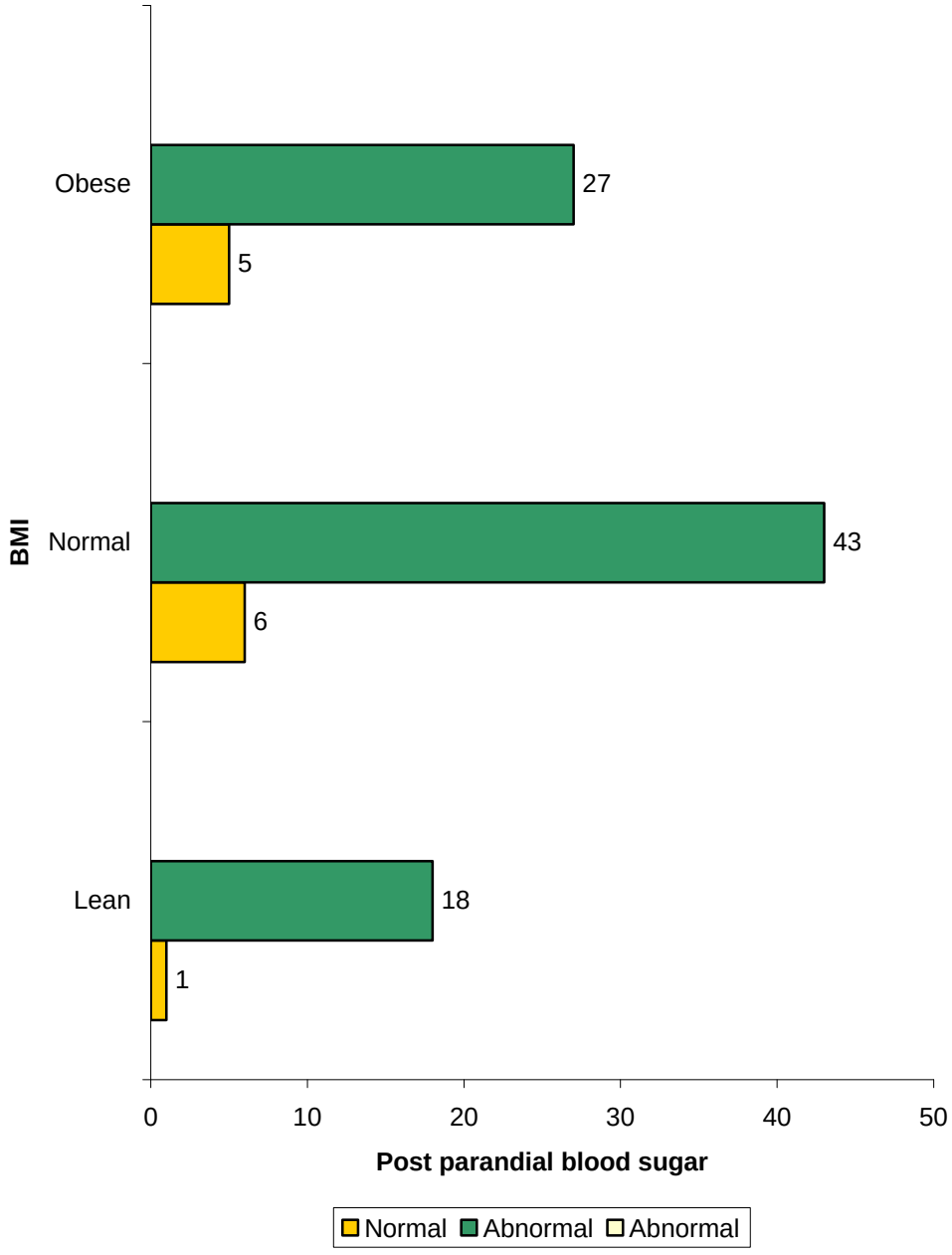


POST PRANDIAL BLOOD SUGAR AND BMI

PP BLOOD SUGAR	LEAN		NORMAL		OBESE	
	NO	%	NO	%	NO	%
NORMAL	1	8.3	6	50.0	5	41.6
ABNORMAL	18	20.5	43	48.9	27	30.7
TOTAL	19		49		32	

There is no statistically significant relationship between PPBS and BMI.

POST PRANDIAL BLOOD SUGAR AND BMI



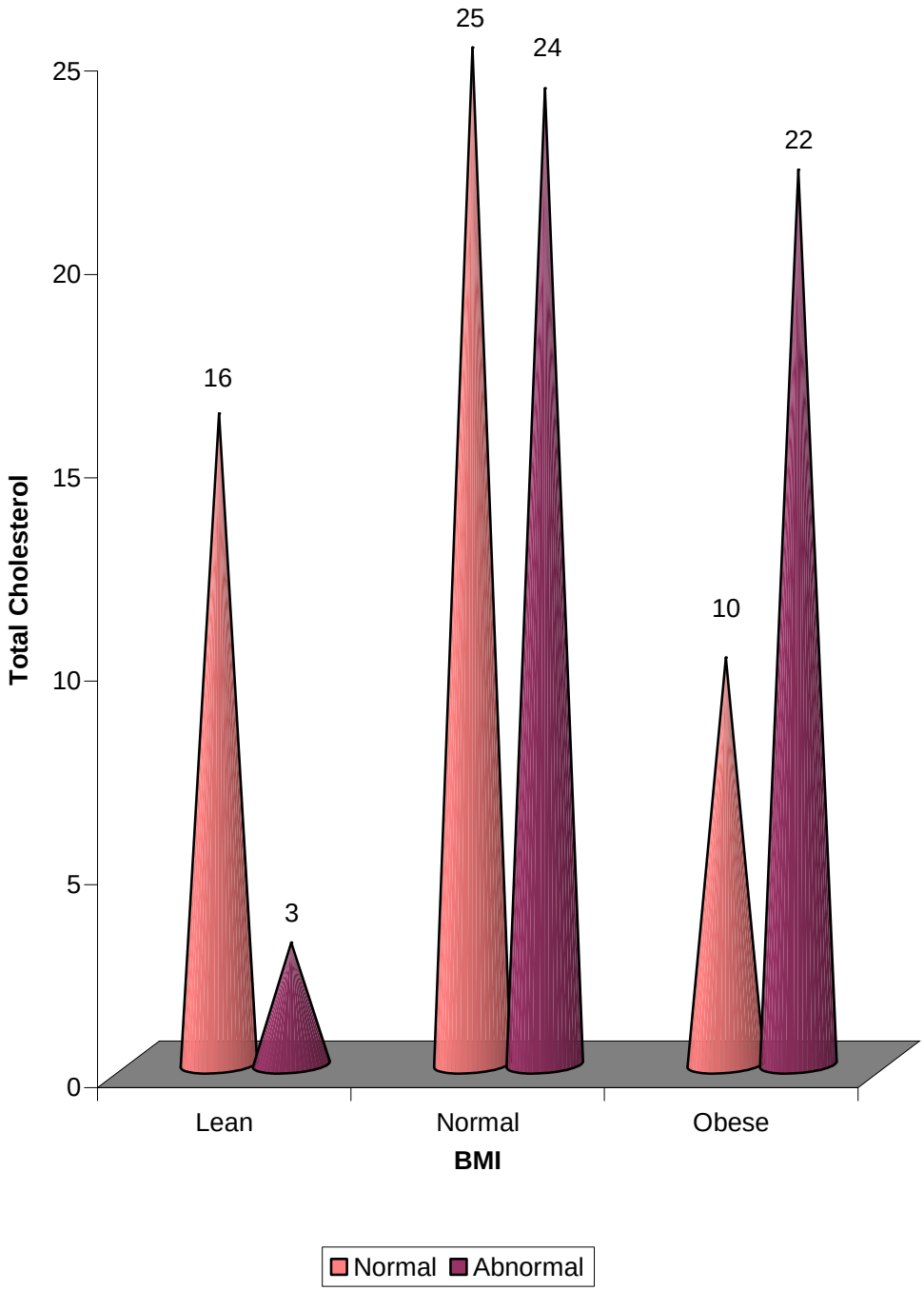
LIPID PROFILE AND BMI

TOTAL CHOLESTEROL	LEAN		NORMAL		OBESE	
	NO	%	NO	%	NO	%
NORMAL	16	31.4	25	49.0	10	19.6
ABNORMAL	3	6.1	24	48.9	22	44.9
TOTAL	19		49		32	

There is statistically significant relationship between total cholesterol and BMI.

‘p’ < 0.001.

LIPID PROFILE AND BMI



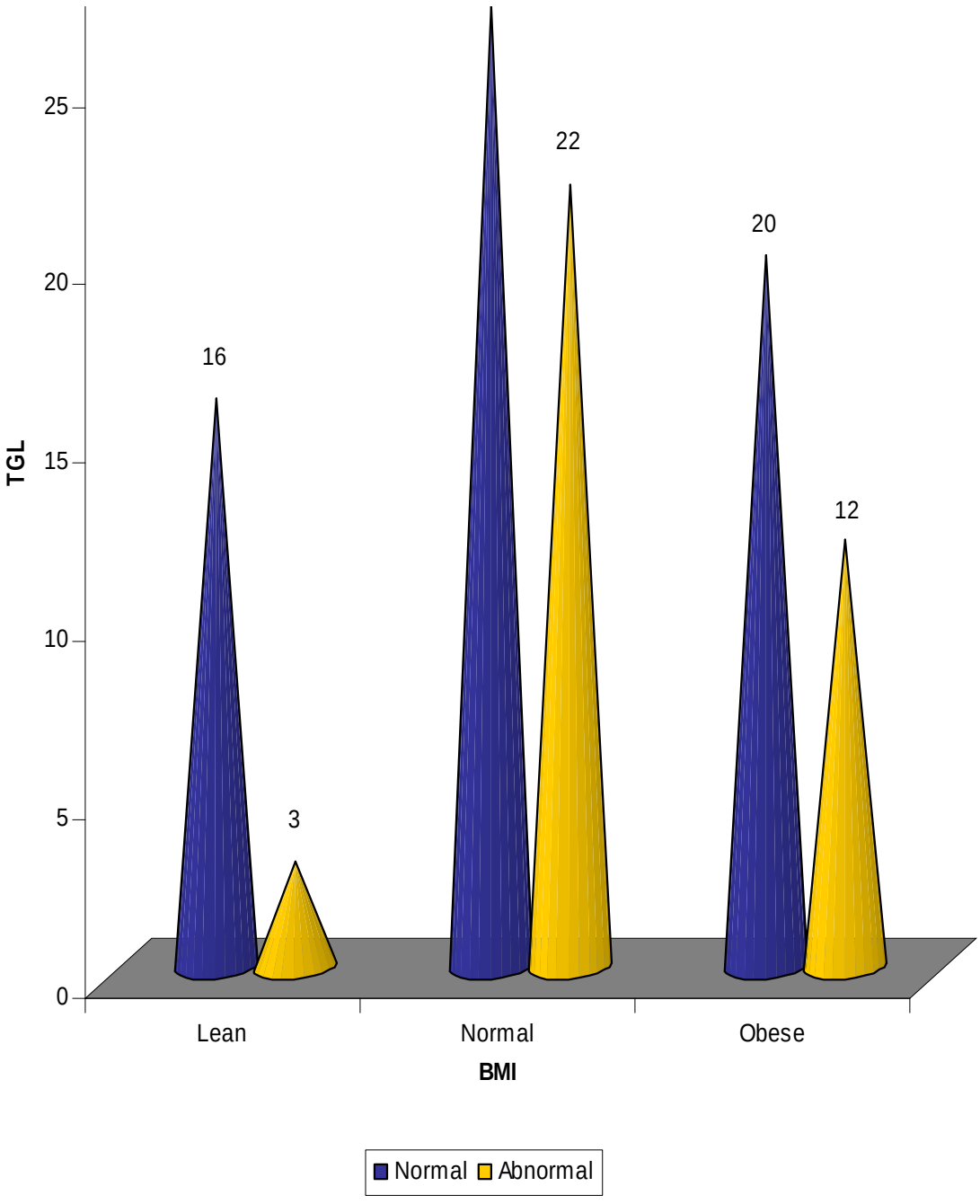
TGL AND BMI

TGL	LEAN		NORMAL		OBESE	
	NO	**%**	**NO**	**%**	**NO**	**%**
NORMAL	16	32.0	27	54.0	20	40.0
ABNORMAL	3	6.0	22	44.0	12	24.0
TOTAL	19		49		32	

There is statistically significant relationship between TGL and BMI.

‘p’ < 0.001

TGL AND BMI


25
20
15
10
5
0
TGL
16
3
22
20
12
Lean
Normal
Obese
BMI
Normal
Abnormal

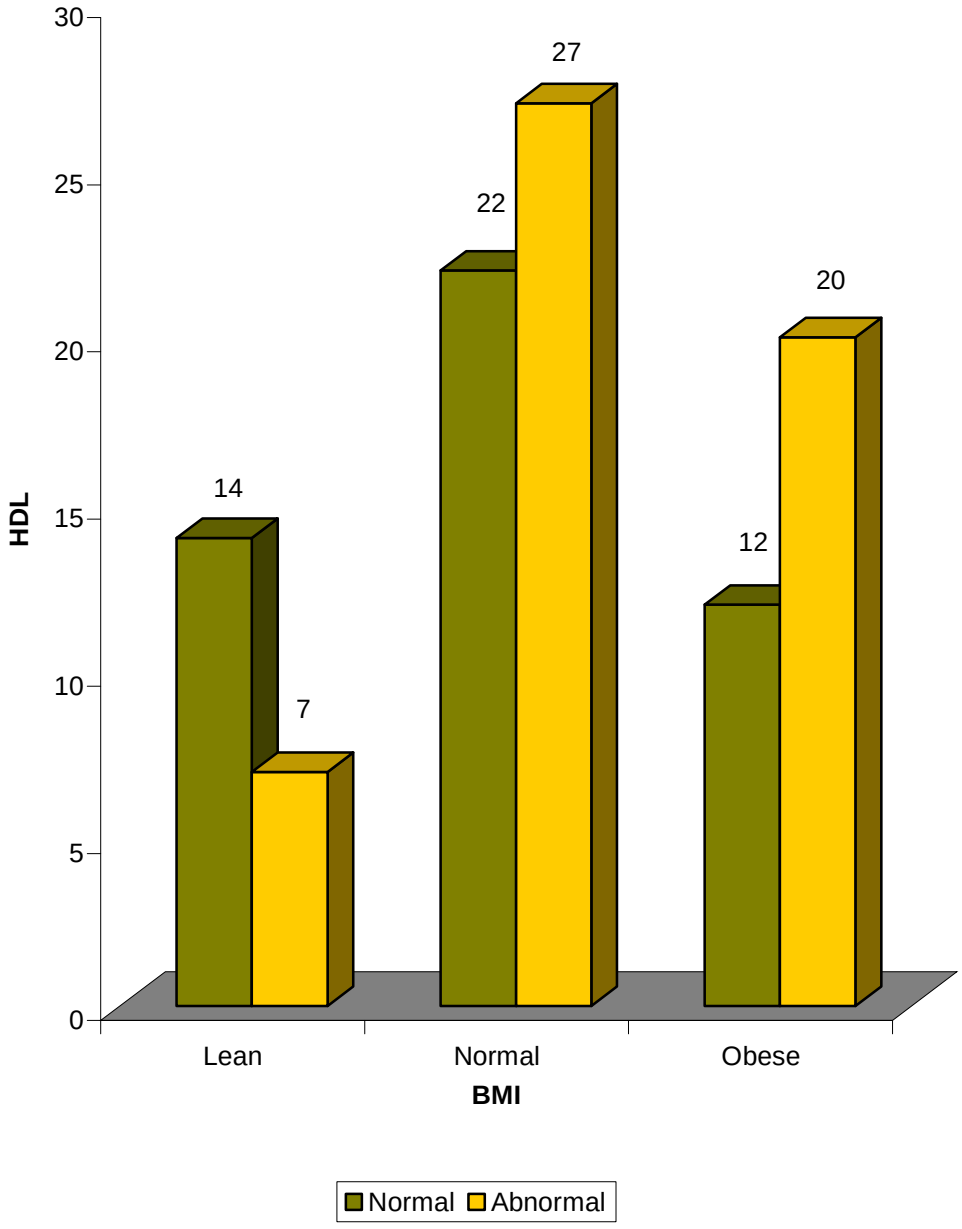
HDL AND BMI

HDL	LEAN		NORMAL		OBESE	
	NO	%	NO	%	NO	%
NORMAL	12	26.1	22	47.8	12	26.1
ABNORMAL	7	12.9	27	50.0	20	37.0
TOTAL	19		49		32	

There is statistically significant relationship between HDL and BMI.

‘p’: 0.01 - significant at 0.05 level

HDL AND BMI


30
25
20
15
10
5
0
HDL
14
7
22
27
12
20
Lean
Normal
Obese
BMI
Normal
Abnormal

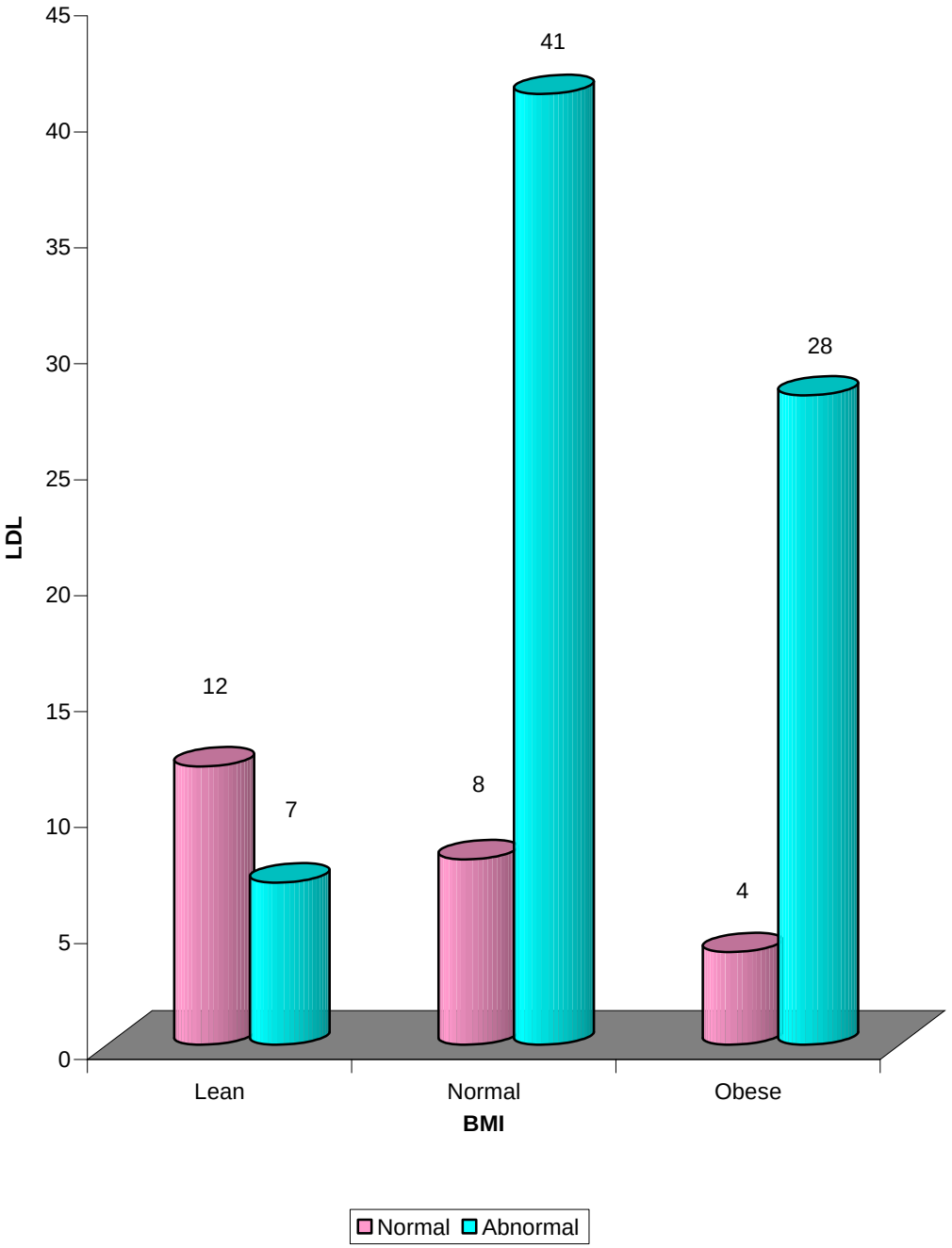
LDL AND BMI

LDL	LEAN		NORMAL		OBESE	
	NO	**%**	**NO**	**%**	**NO**	**%**
NORMAL	12	50.0	8	33.3	4	16.7
ABNORMAL	7	9.2	41	53.9	28	36.8
TOTAL	19		49		32	

There is statistically significant relationship between LDL andBMI.

‘p’ < 0.001

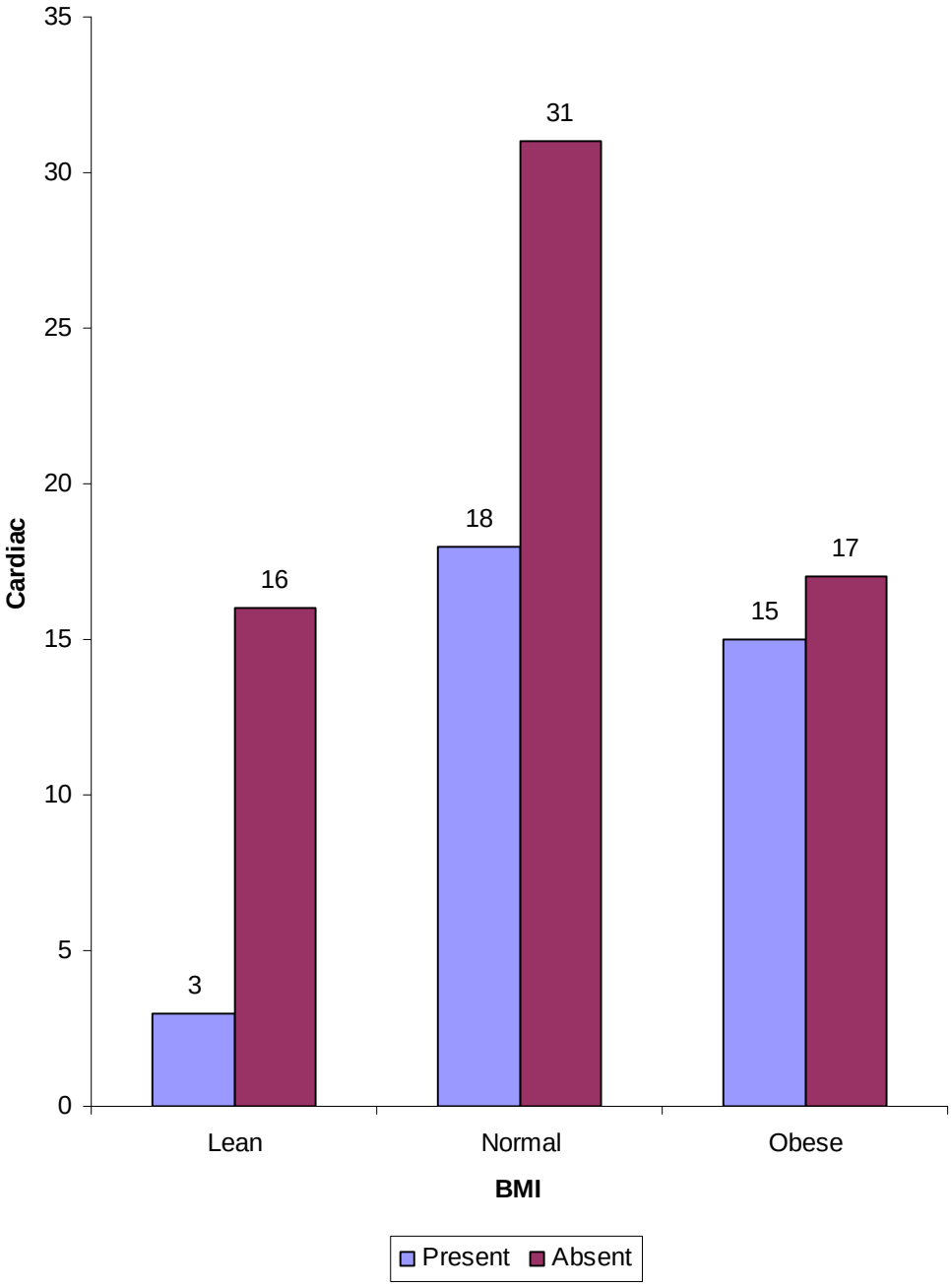
LDL AND BMI


45
40
35
30
25
20
15
10
5
0
LDL
41
28
12
7
8
4
Lean
Normal
Obese
BMI
Normal
Abnormal

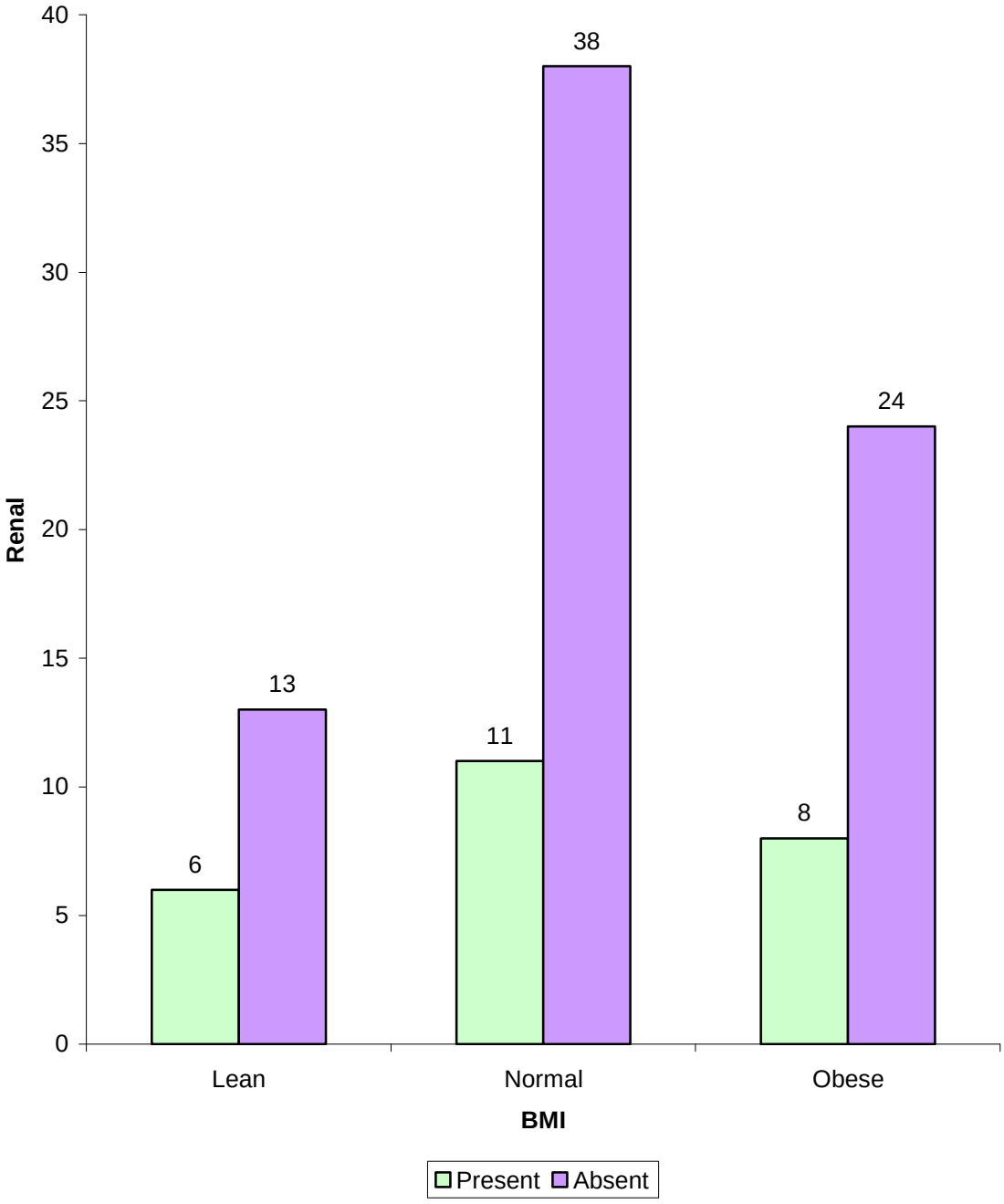
COMPLICATIONS AND BMI

BMI	COMPLICATIONS											
	CARDIAC				RENAL				HYPERTENSION			
	YES		NO		YES		NO		YES		NO	
	NO	%	NO	%	NO	%	NO	%	NO	%	NO	%
L	3	8.3	16	25.0	6	24.0	13	17.3	2	4.6	17	29.8
N	18	50.0	31	48.4	11	44.0	38	50.7	19	44.2	30	52.6
O	15	41.7	17	26.6	8	32.0	24	32.0	22	51.2	10	17.5
p	<0.013 Significant				not significant				<0.001 significant			

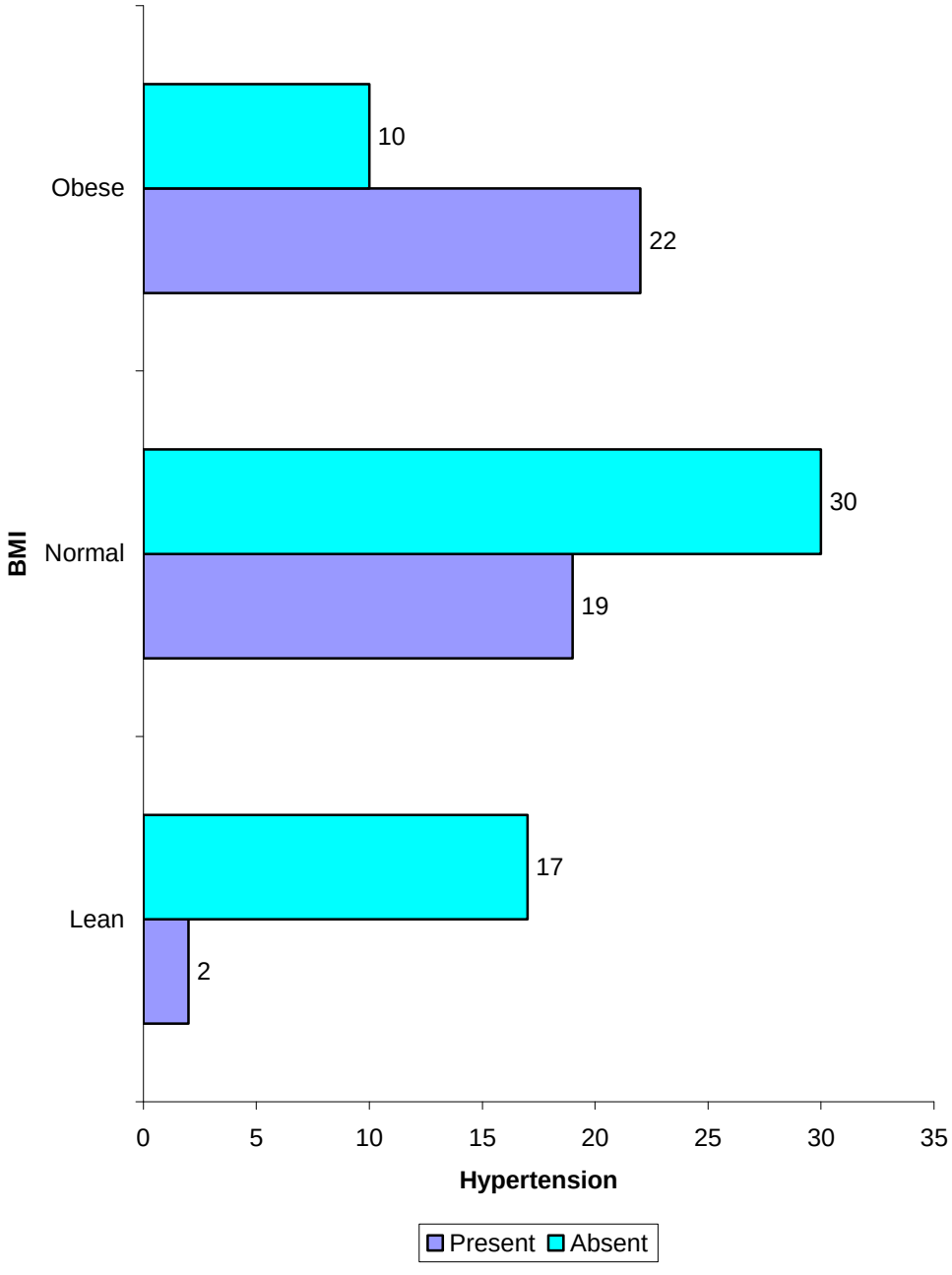
BMI AND CARIDO VASCULAR COMPLICATION



BMI AND RENAL COMPLICATION



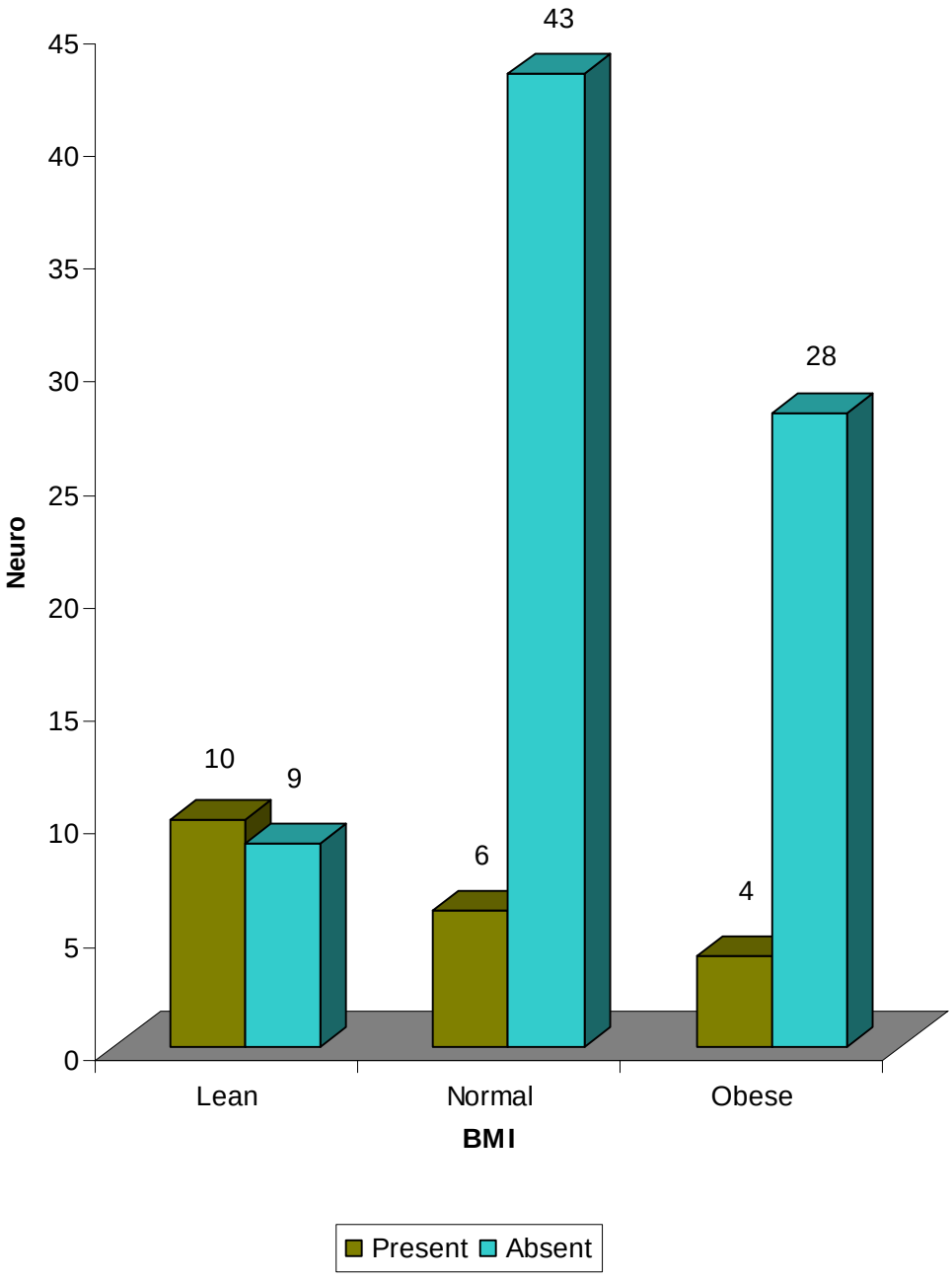
BMI AND HYPERTENSION



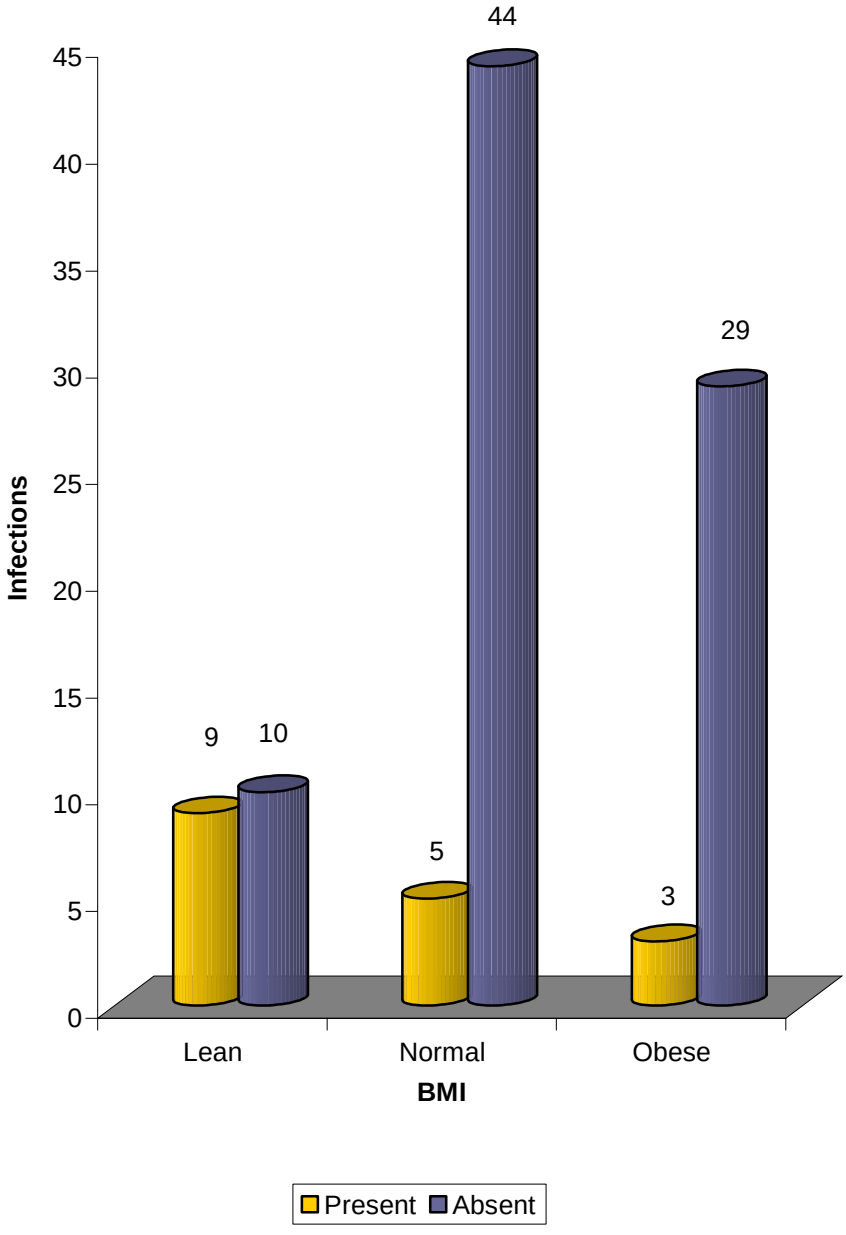
COMPLICATIONS AND BMI

BMI	COMPLICATIONS											
	NEUROPATHY				RETINOPATHY				NFECTIONS			
	YES		NO		YES		NO		YES		NO	
	NO	%	NO	%	NO	%	NO	%	NO	%	NO	%
L	10	45.5	9	11.3	6	35.2	13	15.6	9	52.9	10	13.7
N	6	27.2	43	53.8	7	41.1	42	50.6	5	29.4	44	60.2
O	4	18.1	28	35.0	4	23.5	28	33.7	3	17.6	29	39.7
p	< 0.010 significant				not significant				< 0.001 significant			

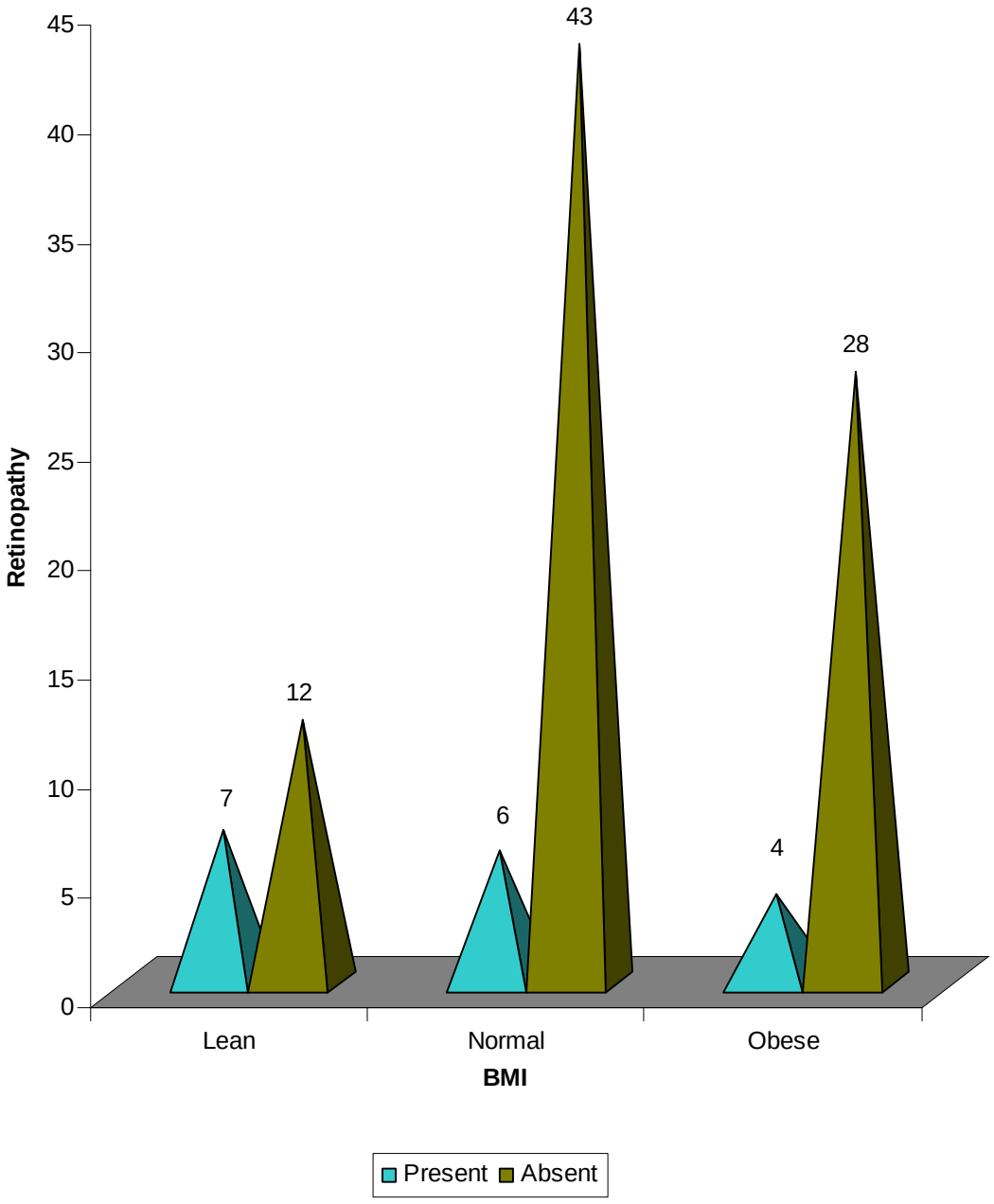
BMI AND NEUROPATHY



BMI AND INFECTIONS



BMI AND RETINOPATHY



DISCUSSION AND ANALYSIS OF RESULTS

DISCUSSION AND ANALYSIS OF RESULTS

"Diabetes Mellitus" is an age old affliction of man and is the most common metabolic disorder all over the world. The incidence of Diabetes is showing alarming rise in developing countries, particularly. In India. India[3] is known as the Diabetic capital of the world. Most of the diabetics in developed countries are obese.

However in India we have a significant number of diabetics who are either normal weight or even under weight[4]. Even though obesity is considered as part of Syndrome X in the pathogenesis of type 2 diabetes, in our study, out of 100 patients only32 patients were obese. Our study included hundred patients. Among **100 patients**, 48 are males and 52 are females. In our study majority of patients, that is 49 patients (49%) belong to normal weight, 32 patients (32%) belong to obese and **19 patients (19%)** belong to **lean body weight**.

1. Age

In our study, we found there is no particular age group for lean diabetics. But 24.5% of lean diabetics belong to 51-60 years of age, 57.1% of normal weight patients between 41-50years,and 55.6 %of obese patients belong to <40 years age group.

2. Sex

There is statistically significant relationship exist in our study between sex and BMI. Slightly higher incidence of female in lean body weight (21.2%) and in normal body weight (51.9%) was observed. In obese there is male preponderance (37.5%)

3. Family History

Family history of diabetes is present only in 9.6% of lean diabetics, in comparison to 35.4% in normal and 54.8% in obese diabetics. So there is lesser incidence of family history among the lean diabetics.

4. Waist Hip Ratio and BMI

There is a linear increase in number of patients having abnormal. Waist Hip ratio with increase in BMI. Among 100 patients studied, 64 patients have abnormal Waist Hip ratio. Among that it is 12.5% in lean, 42.2% in normal and 45.3% in obese type 2 diabetics. Eventhough 19% (19) of diabetics patients are lean based on BMI, 8 among them have abnormal Waist hip ratio. So, **Waist hip ratio is a better indicator than BMI** for assessment of obesity.

5. Complication

Microvascular complications

Among the microvascular complications **neuropathy (45.5%), Retinopathy (35.2%)** are common in **lean diabetics** in our study which are statistically significant. Only 24% of lean diabetics had nephropathy, which is not statistically significant. In normal weight group, incidence of neuropathy, retinopathy and nephropathy are 27.2%, 41.1% and 44% respectively. In obese patients, incidence of neuropathy, retinopathy and nephropathy are 18.1%, 23.5% and 32%respectively.

Macrovascular Complications

Lean diabetics are **less prone** to develop **macrovascular** complications – like hypertension and Ischemic Heart Disease. Incidence of hypertension is 4.6% in lean patients as compared to 44.2% in normal and 51.2%% in obese diabetics. Like wise cardiac complications are low in lean diabetics (8.3%)as compared to 50% in normal and 41.7%in obese diabetics.

Infections

In our study, 52.9% of lean patients with type 2 diabetes presented with infections as compared to 29.4% in normal and 17.6 % in obese

patients. Values are statistically significant- p value < 0.001.Majority of the **lean diabetics** in our study group presented with **infections**.

Glycemic Control

Lean diabetics have more severe hyperglycemia with poor metabolic control. Lean persons have **higher fasting blood sugar** levels than obese and normal weight type 2 diabetes patients. Similarly post prandial values were also high in lean type 2 DM patients. This has been explained by probable **low beta cell reserve** among lean diabetics. So, Lean diabetics are **insulinopenic** and **highly insulin sensitive**.

Lipid Profile

Regarding lipid profile of lean type 2 diabetes patients, all the parameters were **lower** in lean diabetics compared to all other groups. Moreover, lean diabetics have slightly higher HDL value as compared to normal and obese diabetics, which is statistically significant. Also free cholesterol value in lean diabetics are not as high as compared to obese patients. So, lean diabetics have **favorable lipid profile** as compared to normal and obese diabetics.

In contrast to the previous studies, in our study the triglyceride levels were not significantly high in lean diabetics.

Our study has **limitations**, as it was hospital based in the tertiary care setting. Incidence of complications might be higher compared to general population or primary care setting. We did not do HbA1C, insulin level assay, C peptide levels and GAD antibodies in our lean diabetics due to financial constraints. In conclusion, type 2 diabetic patients need not always be obese. Majority (49%) belong to normal weight and significant number (19%) of patients are even lean in our study. Thus, lean body type 2 DM patients appear to be a distinct variety and a great deal of emphasis is to be given on its clinical/biochemical profile and natural history.

COMPARATIVE ANALYSIS

COMPARATIVE ANALYSIS

Our study includes 100 patients with type 2 diabetes. Among them normal weight (49%), obese patients (32%) and lean type 2diabetics (19%) were identified.

But the study

i) Conducted at Manipal by Prabhu Mukhyaprana and Sudha Vidyasagar included 500 type 2 diabetic patients between July 2000 and January 2001.

ii) The study conducted by Gohel Dr. Desai VK at M.P. Shah Medical College, Jamnagar, published in JAPI, Dec 2003 included 75 patients with Type 2 Diabetes Mellitus.

1. Percentage of Lean Body Weight Type 2 DM Population

In our study, Lean Type 2 DM was observed in 19%, as compared to 49% of normal and 32% of obese patients. Study conducted (by Mukhyaprana et al) lean were 7.4% and majority (65%) were of normal weight. Incidence of lean body weight – Diabetes in various Indian studies ranges from 1.6% as in Ramachandran et al. study to as high as 28% as in Tripathi et al.

Mohan et al reported an incidence of 3.5%.

2. Age Group

In our study there is no statistically significant relationship between age and BMI observed.

i) But study conducted by Prabhu et al, mean age of onset of diabetes in lean were 60.34 + 13.5 years.

ii) In Gohel DR. et al study it was between 30-40 years.

3. Sex

In our study, lean type 2 Diabetes patients were slightly higher in female sex (21.2%) which was statistically significant.

i) Study conducted by Prabhu Mukhyaprana M et al observed most lean type 2 DM were males (65% of total lean) type 2 DM which was statistically not significant.

4. Family History

Positive family history was present only in 9.6% of patients with lean body weight type 2 DM as compared to 35.4% in normal weightand 54.8% in obese patients with type 2 DM which were statisticallysignificant.

i) Study conducted by Prabhu Mukhyaprana et al observed positive family history in 45% of lean and 62.6% in normal

body weight diabetics, results were similar to studies by – Banerji et al and Kannan et al studies.

ii) Study conducted by Gohel DR et al observed low incidence of positive family history (20%) in lean as compared to 40% in normal and 44% in obese patients.

5. BMI and WHR – Are they related?

In our study 12.5% of lean diabetes had abnormal Waist Hip Ratio as compared to 42.2%% in normal and 45.3% in obese patients. Waist Hip Ratio had a statistically significant ('p' – 0.001). Relationship with BMI. Previous study conducted at Manipal observed 48% of lean diabetics had abnormal Waist Hip Ratio, stating that significant number of lean diabetics (48%) had abnormal Waist Hip Ratio. The Waist Hip Ratio may thus be a more sensitive indicator of obesity in Indians[25,27].

7. Glycemic Status

In our study, significant proportion of lean persons had higher fasting blood sugar levels than obese patients with Type 2 Diabetes, which was statistically significant (p = -0.112) as compared to normal and obese patients with Type 2 Diabetes.

i) Results were similar to studies done by Kannan et al and Italian Study by Pointoroly et al. This has been explained based on low – beta cell reserve in these patients.

ii) Similar results were also observed in study conducted by Prabhu Mukyaprana et al. Fasting blood sugar was 177.08+105.1.

iii) Postprandial blood sugar values in Lean type 2 DM patients were higher, even though statistically not significant.

8. Lipid Profile

Analysis of lipid profile in our study showed interesting results. Type 2 lean diabetics, had lower incidence of dyslipidemia as compared to all other groups, even though only HDL relationship with BMI was statistically significant. In our study HDL values were slightly higher in lean Diabetics, as compared to normal and obese patients which was statistically significant (p 0.012). Also free cholesterol value in lean diabetics were high as compared to normal weight and obese patients. Triglyceride values in lean diabetics were not very high as compared to normal and obese diabetics. Previous studies by Banerji et al and Das et al had showed slight increase in TGL and HDL in lean diabetics.

Japanese study by Ikeda et al showed no major differences in lipid profile in lean diabetics, irrespective of glycemic status.

9. Complications

In our study, increased incidence of **microvascular** complications like neuropathy, retinopathy were observed in lean diabetics which is statistically significant. 47.3% of lean patients had neuropathy as a presenting feature as compared to 31.5% in normal and 21.1% in obese patients with a 'p' value of 0.010 (significant). Retinopathy also increased in lean type 2 Diabetics with 35.2% in lean, 41.1%in normal and 23.5% in obese patients though it is not statistically significant. In our study nephropathy was observed only in 24% of lean patients as compared to 44%in normal and 32% of obese type 2 diabetics, which is not statistically significant. Study conducted at Manipal showed microvascular complications were similar in all the 3 groups. **Macrovascular complications** like HT, IHD were less in lean[17] diabetics as compared to other groups. In our study, the incidence of hypertension was 4.6% in lean as compared to 44.2% in Normal and 51.2 % in Obese. Incidence of IHD was low in the lean as compared to normal and Obese. Incidence of hypertension was 8.9% and IHD 10.2% in Nigam et al

study. In Manipal study the incidence of IHD was only 2.7% among lean and HT in 16.7% of lean diabetics.

Infections

In our study 52.9% of lean patients with type 2 diabetes presented with infections as compared to 29.4% in normal and 17.6%in obese patients. Values were specifically significant also. ('p' value = < 0.001). Mohan et al[14] reported increased prevalence of retinopathy, neuropathy and nephropathy in lean diabetics. Peripheral neuropathy was the commonest presenting complication among lean diabetics in a study by Das et al. Peripheral neuropathy and **infections** were the commonest presenting clinical features in lean diabetics observed in study conducted by Gohel et al.

SUMMARY

SUMMARY

- Total Number of patients studied – 100. Out of 100 patients, 48 were males and remaining 52 were females.
- Number of lean type 2 DM Patients were 19. Among them 44% were Males and 56% were Females.
- Number of normal weight type 2 diabetics were 49. Among them 45% were males and 55% were females.
- Number of obese type 2 diabetics patients were 32. Among them 56% were males and 44% were females.
- Most of diabetics in our population (52%) had normal body weight. Lean type 2 Diabetics form a significant number (19%).
- Low incidence of positive family history in lean type 2 diabetics (9.6%) was observed.
- Peripheral neuropathy (47.3%), Retinopathy (35.2%) and infections (52.9%%) were the major presenting clinical complications in lean diabetics.
- Most risk factors of atherosclerosis and CAD are less prevalent in lean type 2 diabetes (Normal HDL, and total cholesterol on lower side).

CONCLUSION

CONCLUSION

a. Majority of type 2 diabetes patients in our population are having **normal weight (49%)** and **lean body weight contributes to 19%**

b. Lean diabetics have **more severe hyperglycemia** and poor metabolic control. They are more prone for **microvascular complications** like neuropathy and retinopathy.

c. **Early treatment** with **insulin** in lean type 2 diabetics is mandatory to achieve **good glycemic control** and to prevent future complications.

APPENDIX

BIBLIOGRAPHY

BIBLIOGRAPHY

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PROFORMA

PROFORMA

CLINICAL PROFILE OF LEAN BODY WEIGHT TYPE 2 DM PATIENTS IN COMPARISON WITH OBESE AND NON-OBESE TYPE 2 DM PATIENTS

Date of Registration No.

Name /Age /Sex

Address

Occupation

Sedentary

Active

New case/ Already treated case How long

PRESENTING SYMPTOMS AT THE TIME OF DETECTION

Symptoms	**YES**	**NO**
Polyuria		
Polydypsia		
Polyphagia		
Tiredness		
Weight loss		
Blurring of vision		

Itching

Vomiting

Abdominal pain

Constipation

Nocturnal Diarrhoea

Numbness

Pruritus vulvae

Sexual Dysfunction

Balanitis

joint / body pain

Ulcer

Previous treatment: Insulin /tablets

Diet

Alternative

Previous illness: M.I

HT/ Operations

Jaundice/PT

Family History of Diabetes: Yes/no

Personal History

Smoking	Yes/no
Alcohol	Yes/no
Vegetarian	Yes/no
Non- veg	Yes/no

Examination

General: Acanthosis Nigricans

Xanthoma / Xanthelasma

Thyroid swelling

BP: Lying/Standing

Pulse

Anthropometry

Height (cm)/weight(kg)

Under Wt. (Lean)/Ideal wt/ Obese

BMI

Waist / hip ratio (WHR)

System

CVS

Respiratory

Abdomen

CNS

Opthalmological

Investigations

Urine - Albumin

Sugar

Deposits

Blood sugar Fasting

Post prandial

Blood urea

Sr. creatinine

Lipid profile

TCL

LDL

HDL

VLDL

TGL

ECG

ASSESMENT

1. Type 2 DM

lean

Normal wt

Obese

2. Family history: Present/ Absent

3. Metabolic: Fasting Hyperglycemia Yes/No

4. vascular:

IHD	Yes/no
PVD	Yes/no
Retinopathy	Yes/no
Nephropathy	Yes/no
Neuropathy	Yes/no
Infections	Yes/no
Others (specify)	Yes/No

MASTER CHART

MASTER CHART

S. NO	NAME	TYPE	AGE	SEX	BMI		WHR		FAMILY HISTORY	FBS	PPBS	TC	TGL	HDL	LDL	VLDL	CVD	NEPHRO	NEURO	RETINO	INFECTION	HT	SBP	DBP
1.	Selvamani	N	60	1	24.12	2	0.78	1	2	248	480	248	193	45	165	38	2	2	2	2	2	1	150	100
2.	Gnanamal	N	54	2	20.5	2	0.9	2	2	191	335	225	209	67	117	41	1	2	2	2	2	2	110	80
3.	Meenambal	N	50	2	23.6	2	0.8	2	2	131	206	203	242	86	69	48	2	2	1	2	2	1	150	100
4.	Karuppan	L	55	1	15..4	1	0.84	1	2	114	278	167	218	78	70	42	2	1	2	2	1	2	120	80
5.	Annavi	O	36	1	30.4	3	0.97	2	1	145	347	256	140	44	187	25	1	1	2	2	2	2	110	80
6.	Jameela	L	52	2	18.04	1	0.91	2	2	239	476	190	96	62	109	19	2	1	1	2	1	1	100	70
7.	Murugan	L	55	1	16.89	1	0.87	1	2	207	389	166	170	68	73	25	2	2	1	2	1	2	126	80
8.	Danushkodi	O	55	1	30.43	3	0.9	1	1	107	214	204	160	36	136	32	2	2	2	2	2	1	160	100
9.	Mumtaz	O	45	1	30.36	3	0.97	2	1	336	460	190	153	38	122	30	2	2	2	1	2	1	160	90
10.	Mary	L	60	2	17.85	1	0.91	2	2	103	209	166	86	62	87	17	2	1	2	2	1	2	120	78
11.	Samikkanu	N	50	1	19.11	2	0.87	1	2	300	428	210	137	41	143	27	2	2	1	2	2	2	120	84
12.	Meenambigai	O	43	2	32.89	3	0.81	2	1	108	215	287	325	80	145	65	2	2	2	2	2	1	150	90
13.	Vasuderan	O	40	1	30.79	3	1	2	1	192	341	216	186	42	37	137	2	1	2	2	2	1	150	100
14.	Shahul Hameed	O	38	1	32.04	3	0.98	2	1	235	375	206	193	45	143	38	2	2	2	2	2	1	140	90
15.	Jayanthi	O	47	2	30.33	3	0.95	2	2	106	300	260	96	48	193	19	2	2	1	2	1	1	150	86
16.	Nazira	L	52	2	18.49	1	0.91	2	2	312	418	226	146	47	150	29	1	2	1	1	2	2	120	80

17.	Thangavel	O	50	1	30.47	3	0.96	2	1	128	298	187	89	51	119	17	2	1	2	1	2	2	110	84
18.	Chandra	N	45	2	23.63	2	0.93	2	2	132	246	186	118	47	116	23	2	2	1	2	2	2	110	70
19.	Selvi	O	38	2	30.32	3	0.9	2	1	271	446	222	86	56	149	17	2	1	1	2	2	2	100	70
20.	Muthukutti	L	55	1	18.36	1	0.91	1	2	366	490	201	186	66	114	21	2	1	2	1	2	2	100	74
21.	Murugaiyan	O	62	2	30.45	3	0.97	2	2	133	270	244	102	56	168	20	2	2	1	2	2	1	140	100
22.	Chinnaponnu	N	60	2	21.09	2	0.85	2	2	212	360	124	198	47	35	39	2	1	2	2	2	1	140	100
23.	Chellammal	N	52	2	22.01	2	0.88	2	2	139	232	209	115	61	144	116	2	1	2	2	2	2	120	80
24.	Amutha	N	45	2	21.17	2	0.94	2	2	246	486	250	162	74	144	116	2	2	1	2	2	2	120	70
25.	Babulal	N	60	1	21.3	2	0.93	1	1	250	452	178	348	44	65	69	1	2	2	2	2	1	170	96
26.	Gnanasekaran	L	65	1	18.08	1	0.88	1	1	318	499	203	109	35	126	22	1	2	1	2	1	2	120	80
27.	Selvaraj	N	48	1	19.53	2	0.97	2	1	210	337	276	155	80	165	31	2	2	2	1	2	2	110	80
28.	Kamarchi	N	58	2	20.5	2	0.83	2	2	233	388	164	145	89	29	96	2	1	2	2	2	2	120	76
29.	Chinnasamy	N	64	1	21.09	2	0.89	1	2	160	324	167	55	39	117	11	2	1	2	2	2	2	120	70
30.	Subbulakshmi	O	56	2	37.02	3	1	2	1	210	498	185	255	42	51	92	1	2	2	2	2	1	160	100
31.	Shyamala	N	58	2	22.5	2	0.98	2	2	188	346	250	172	40	186	34	2	2	2	1	2	2	120	80
32.	Rubella mary	N	64	2	19.13	2	0.89	2	2	140	299	220	141	38	154	28	2	2	2	2	2	1	150	90
33.	Sarfunnissa	O	53	2	30.47	3	0.92	2	1	309	430	246	81	38	198	16	2	2	2	1	2	1	140	96
34.	Angayee	N	58	2	21.77	2	0.89	2	1	176	298	192	138	36	125	27	1	2	2	2	2	1	150	90
35.	Jovan	O	58	1	33.91	3	0.92	2	1	185	399	223	123	45	153	24	1	2	2	2	2	1	150	96

36.	Chockalingam	O	55	1	30.45	3	0.97	2	2	129	230	202	68	64	125	13	2	2	2	2	2	1	150	100
37.	Rajalingam	O	58	1	35.15	3	0.91	1	1	134	286	159	42	64	42	13	2	1	2	2	2	1	160	100
38.	Chinnammal	N	54	2	23.78	2	0.82	2	2	180	366	252	81	38	198	16	2	2	2	2	2	1	120	80
39.	Rangasamy	L	60	1	18.49	1	0.81	1	2	356	444	168	60	48	108	12	2	1	2	2	2	2	110	74
40.	Annabakiyam	N	64	2	19.59	2	0.9	2	2	169	350	223	58	40	172	11	1	2	2	2	2	2	120	80
41.	Gnanaprakasam	L	60	1	18.22	1	0.85	1	2	204	386	194	96	68	107	19	2	1	2	2	2	2	120	70
42.	Pappa	L	58	2	17.36	1	0.86	2	2	210	348	198	104	58	118	22	2	2	2	1	2	2	110	70
43.	Saroja	N	60	2	23.31	2	0.81	2	2	145	304	262	148	51	183	29	2	2	2	1	2	1	140	94
44.	Kalaivani	L	48	2	18.17	1	0.85	2	1	176	281	173	98	46	108	19	2	2	1	2	2	2	110	70
45.	Abdual kadhar	O	57	1	22.49	3	0.93	1	1	280	364	225	162	38	155	32	1	2	2	2	2	1	160	96
46.	Mohammed Ali	N	62	1	24.03	1	0.92	1	1	160	327	246	148	45	172	29	1	2	2	2	2	1	134	98
47.	Gandhimathi	L	58	2	17.31	1	0.84	2	2	208	430	138	98	35	82	19	2	2	1	2	2	2	110	70
48.	Sabeera	N	60	2	23.78	2	0.81	2	2	204	378	152	208	25	86	41	2	1	2	2	2	2	120	80
49.	Parvathi	O	55	2	30.12	3	0.92	2	2	190	470	188	105	29	140	21	1	1	2	2	2	1	150	100
50.	Senthamilselvan	N	38	1	24.8	2	0.97	2	2	392	403	182	146	28	125	29	2	2	2	2	1	1	160	100
51.	Raman	O	62	1	31.2	3	0.97	2	2	192	332	202	97	43	140	19	1	2	2	2	2	2	120	80
52.	Govindharajan	N	65	1	19.48	2	0.86	1	2	176	223	184	97	28	137	19	2	1	1	2	2	2	120	80
53.	Deepa	N	34	2	20.81	2	0.85	2	1	198	306	174	95	30	125	19	2	2	2	2	1	2	110	84
54.	Aminabeevi	N	56	2	20.44	2	0.83	2	2	190	384	170	97	29	128	19	2	1	2	2	1	2	100	70

55.	Fathima	O	56	2	36.44	3	0.92	2	1	192	340	175	95	44	112	19	1	1	1	2	2	1	160	100
56.	Maruthai	L	54	1	18.49	1	0.88	1	2	196	364	161	165	48	84	29	2	2	1	1	2	2	110	60
57.	Adiakalaraj	N	60	1	23.81	2	0.95	1	2	160	386	225	106	32	171	22	1	2	2	2	2	1	150	100
58.	Rengasamy	N	50	1	19.83	2	0.93	1	2	280	392	201	145	42	132	29	1	2	1	2	1	2	100	70
59.	Saradha	N	52	2	22.19	2	0.91	2	2	184	402	256	178	43	178	45	1	2	2	2	2	1	130	96
60.	Kulanthaisamy	O	64	2	33.78	3	0.97	2	2	108	288	286	164	38	216	32	1	2	2	2	2	2	120	80
61.	Abdulla Sheriff	O	62	1	30.22	3	0.93	1	2	134	268	280	162	43	208	32	1	2	1	2	2	2	110	78
62.	Haneef Beevi	N	63	2	22.35	2	0.88	2	1	206	392	200	162	52	118	32	2	2	2	1	2	2	100	70
63.	Chellammal	L	55	2	18.37	1	0.78	2	2	284	262	167	184	68	62	37	2	2	1	2	1	2	120	70
64.	Mari susairaj	O	54	2	30.13	3	0.89	2	2	114	338	266	188	42	186	38	1	2	2	2	2	1	150	90
65.	Rasayee	N	60	2	20.27	2	0.9	2	2	182	449	165	152	54	85	30	2	1	2	2	2	2	100	60
66.	Mookammal	N	65	2	20.54	2	0.91	2	1	176	281	184	142	68	88	28	2	2	1	1	2	2	90	60
67.	Shaitha	N	52	2	20.49	2	0.89	2	1	212	438	184	82	34	134	16	1	2	2	2	2	1	160	94
68.	Bommakka	N	58	2	24.92	2	0.96	2	2	138	207	184	99	45	119	20	2	2	2	2	2	1	150	100
69.	Geetha	L	57	2	18.31	1	0.79	2	2	196	292	153	173	36	93	24	2	2	2	1	2	2	110	80
70.	Paulraj	O	65	1	33.33	3	1	2	2	179	232	226	168	42	155	29	2	2	1	2	1	2	100	60
71.	Anjammal	O	60	2	30.04	3	0.88	2	2	308	410	230	172	42	156	32	2	2	1	2	2	1	140	94
72.	Kannaiyan	L	64	1	18.43	1	0.88	1	2	310	415	166	114	68	75	23	2	2	1	2	1	2	100	60
73.	Balakrishnan	N	65	1	22.15	2	0.92	1	2	280	394	204	158	41	133	32	1	2	2	2	2	2	120	84

74.	Noorjehan	N	59	2	22.03	2	0.79	2	2	238	400	198	166	42	123	33	2	2	1	2	2	2	100	70
75.	Chinnan	O	58	1	30.42	3	0.93	1	2	156	340	188	177	42	113	33	1	2	1	2	2	2	120	80
76.	Khairunnissa	L	58	2	18.26	1	0.79	1	2	258	324	168	172	44	90	34	2	2	1	2	1	2	120	64
77.	Mohan	N	55	1	20.95	2	0.86	1	2	168	336	220	168	40	156	34	1	1	2	2	2	1	160	110
78.	Madhavan	O	59	1	31.89	3	0.97	2	2	210	450	282	178	30	217	35	1	2	2	2	2	1	140	100
79.	Nallammal	L	45	2	16.82	1	0.85	2	2	270	396	166	200	64	62	40	2	2	1	1	2	2	100	70
80.	Ganesan	O	64	1	30.1	3	0.97	2	2	180	306	174	124	30	122	32	1	2	2	2	2	1	150	90
81.	Mangalam	L	65	2	17.78	1	0.79	1	2	250	486	148	164	56	68	24	2	2	2	2	1	2	100	68
82.	Palanisamy	N	58	1	21.25	2	0.88	1	1	156	395	167	242	45	100	22	1	2	2	2	2	2	110	84
83.	Saravanan	N	63	1	22.14	2	0.9	1	1	180	326	200	176	45	120	35	2	2	2	2	1	2	120	80
84.	Jamesha	O	62	2	30.44	3	0.89	2	1	178	388	288	196	35	214	39	1	2	2	2	2	1	170	100
85.	Saminathan	N	58	1	20.32	2	0.92	1	2	104	390	186	172	43	111	32	2	2	2	1	2	2	110	70
86.	Raja	N	56	1	19.14	2	0.91	1	2	240	401	176	101	34	102	20	2	1	2	2	2	1	148	90
87.	Arumugam	N	65	1	22.19	2	0.89	1	2	150	413	202	155	42	127	33	1	2	2	2	2	2	110	80
88.	Sundharamoorthi	N	38	1	25	2	0.9	1	1	126	224	172	180	42	104	36	1	2	2	2	2	1	140	90
89.	Murugesan	N	45	1	30.47	3	0.97	2	2	136	240	182	166	38	111	33	2	1	2	2	2	1	150	100
90.	Petchiammal	O	62	2	30.79	3	0.93	2	2	188	256	282	188	28	117	37	1	2	2	2	2	2	110	70
91.	Shajehan	O	39	1	33.05	3	0.95	2	1	165	310	236	170	32	170	34	2	2	2	2	2	1	160	90
92.	Mahmoodha	O	68	2	35.49	3	1	2	1	192	400	198	166	42	91	33	2	2	1	1	2	2	120	80

93.	Mariyasusai	O	65	1	30.47	3	0.95	2	2	174	388	204	164	32	137	35	2	2	2	2	1	2	100	70
94.	Kuppammal	N	60	2	22.1	2	0.79	2	2	186	362	204	137	44	136	24	1	2	2	2	2	1	140	90
95.	Susheela	N	55	2	23.44	2	0.79	1	2	92	260	216	156	45	140	31	2	2	1	1	2	2	100	60
96.	Abidha	N	41	2	22.14	2	0.79	2	2	262	436	188	160	40	116	32	2	1	2	2	2	2	110	60
97.	Padma	N	62	1	21.78	2	0.91	1	2	196	278	168	140	42	90	28	1	2	2	2	2	1	140	90
98.	Neelambal	N	40	2	23.81	2	0.79	1	1	128	391	184	166	42	109	33	2	2	2	1	2	2	120	80
99.	Manikandan	N	65	1	20.44	2	0.79	1	2	192	400	188	110	32	134	22	2	1	2	2	2	2	120	70
100.	Gopalan	N	62	1	23.12	2	0.79	1	2	204	382	180	117	34	125	21	1	2	2	2	2	2	120	70

Sex: 1-Male
2-Female

BMI: L-Lean(1)
N-Normal weight (2)
O-Obese (3)

Family History: 1-Yes
2-No

Complication: 1-Yes
2-No

ABBREVIATIONS

BMI	-	Body Mass Index
WHR	-	Waist Hip Ratio
DM	-	Diabetes Mellitus
MODY	-	Maturity onset diabetes of the young
CHO	-	Carbohydrate
LADA	-	Latent Auto immune diabetes in aduls
IGT	-	Impaired glucose tolerance
TC	-	Total Cholesterol
TGL	-	Triglycerides
HDL	-	High Density Lipoproteins
LDL	-	Low Density Lipoprotein
VLDL	-	Very Low Density Lipoprotein
FPG	-	Fasting Plasma Glucose
PPG	-	Postprandial Plasma Glucose
AGE	-	Advance Glycosylation end product
ADA	-	American Diabetes Association
NGSP	-	National Glycohemoglobin Standardization programme.
DCCT	-	Diabetes Complication Control Trial